TRANSACTIONS

OF THE

TWENTY-EIGHTH ANNUAL MEETING

OF THE

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

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TABLE OF CONTENTS.

ANNUAL MEETING.

President's Address..... 1

Papers.

Some Phases of Esophageal Stenosis. Richmond McKinney, M. D.. 8
Precautions in Using Local Anesthesia with the Report of a Death. Curtis C. Eves, M. D..... 14
Some Remarks on Otitic Malignancy; Report of Two Cases. Hemorrhage Controlled with a Wooden Plug. J. Clarence Keeler, M. D..... 25
The Intimate Lymphatics of the Trachea. Jerome Frank Strauss, M. D..... 35
I. Two Cases of Mastoiditis, Sinus Thrombosis, Facial Paralysis on Opposite side, Double Choked Disc; Recovery.
II. Three Cases of Meningitis Developing Six Weeks to Four Months After Mastoidectomy.
III. Rupture of Carotid Artery: Result of Chronic Mastoidectomy. Wesley C. Bowers, M. D..... 46
A Contribution to the Study of Brain Abscess. J. K. Milne Dickie, M. D..... 58
The Histopathology and Histogenesis of Benign Growths of the Nose and Accessory Cavities. Harry L. Baum, M. D..... 69
Latent Empyema of the Maxillary Antrum. W. G. Harrison, M. D.. 86
A Septic Type of Temperature Not Referable to the Ear in Cases of Acute Suppurative Otitis Media. Harold I. Lillie, M. D... 93
Audiometric Methods and Their Applications. E. P. Fowler, M. D. and Mr. R. L. Wegel, Research Laboratory of the American Telephone and Telegraph Co. and Western Electric Co..... 98
Case of Polyarthritis Complicating Mastoiditis. Operation. Recovery. Dunbar Roy, M. D..... 133
The Upper Respiratory Tract in Granite Dust Inhalation. D. C. Jarvis, M. D..... 139
Reflexes from the Respiratory Tract. Holmes C. Jackson, M. D... 147
Some Points in the Comparative Anatomy of the Nose and the Accessory Sinuses Which Account for the Variations in These Structures in Man. J. M. Ingersoll, M. D..... 162
Fatalities Following Operations Upon the Nose and Throat Not Dependent Upon Anesthesia. A Study of Three Hundred and Thirty-Two Hitherto Unreported Cases. Hanau W. Loeb, M. D..... 168
Aural Exostosis. F. A. Burton, M. D..... 191

The Present Status of Skiagraphic Interpretation As An Adjunct in the Diagnosis of Catarrhal Affections of the Accessory Sinuses. Ross Hall Skillern, M. D..... 214
Complications of Paranasal Sinus Disease in Infants and Young Children. L. W. Dean, M. D..... 221
The Importance (1) of the Absence of the Reactibility of the Vertical Canals of Both Ears to Stimulation By the Cold Caloric in the Diagnosis of Increased Intracranial Pressure of the Posterior Fossa, and (2) of the Reduction of the Duration of the Nystagmus from Turning in Diffuse Legions of the Cerebrospinal System Pathways Over the Cerebellar Cortex (As in Protective Meningitis and Linear Fractures of the Skull of the Posterior Fossa). Wells P. Eagleton, M. D. 231
Epidemic Encephalitis Auditory and Vestibular Lesions. Isidore Friesner, M. D..... 285
General Considerations in Inflammatory Diseases of the Nasal Accessory Sinuses. Duncan MacPherson, M. D..... 291

MEETING OF THE EASTERN SECTION.

Papers.

Some Remarks on the Indication for the Simple Mastoid Operation in the Adult. John B. Rae, M. D..... 305
Treatment of Acute Sinus Infections. Lee M. Hurd, M. D..... 311

MEETING OF MIDDLE SECTION.

Papers.

Actinomycosis of the Tongue. Gordon B. New and Fred M. Figi, M. D..... 315
X-Ray Diagnosis of Unusual Laryngo-Tracheal and Esophageal Conditions and Diseases. Samuel Iglauer, M. D..... 322

MEETING OF THE SOUTHERN SECTION.

Papers.

Items of Progress From Last Year's Literature of Otorhinolaryngology. H. O. Reik, M. D..... 236
A Method of Demonstrating the Surgical Anatomy of the Mastoid. Jesse Wright Downey, M. D..... 335
Subcutaneous Empysema of the Neck and Chest Following Tonsellectomy in an Epileptic. Recovery. Sylvan Rosenheim, M. D..... 337
Adam Politizer-In Memoriam. Harry Friedenwald, M. D..... 342

MEETING OF THE WESTERN SECTION.

Papers.

The Tonus Effects of the Cristae and of the Maculae. S. S. Maxwell, Dept. of Physiology of the University of California. 344

Certain General Conditions Underlying the Care of Acute Mastoiditis. Eugene Richards Lewis, M. D..... 351

MEETING OF THE MID-WESTERN SECTION.

Papers.

Difficulties in Interpretation of Tests of the Vestibular Mechanism. Frank L. Dennis, M. D..... 356
Demonstration of Cases. Wm. C. Bane, M. D..... 382
Gastrostomy in Laryngeal Tuberculosis. Claude E. Cooper, M. D..... 383

MINUTES AND REPORTS.

Business Meeting..... 391
Executive Session..... 395
Report of Section Meetings..... 395
Scientific Session..... 397
Report of Secretary..... 398
Report of Treasurer..... 399
Report of Auditing Committee..... 401
Report of Nominating Committee..... 402
Miscellaneous Business..... 402
Board of Directors..... 403
Fellows..... 405
Officers..... 416

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Held in Washington, May 1st, 2nd and 3rd, 1922.

ADDRESS OF THE PRESIDENT.

By LEWIS A. COFFIN, M. D., New York City.

Fellow Members of the American Laryngological, Rhinological and Otological Society: .

The advancement of any art or science keeps pace with an army of men who are proceeding shoulder to shoulder on a never ending relay march; the advance leaders of the column getting glimpses of the fair and ever inviting country of the unknown through the rifted, cloudy curtain separating it from the traveled, conquered, yet beloved land of the known.

Fortunately the cloudy veil appears to recede at such a rate that no member of the advancing ranks shall ever penetrate, to become possessed or acquainted with the great beyond; yet making it possible that many of the beautiful spots once revealed through the rifts may be reached, to become, as it were, relay stations, where the joyous traveler receives new energy, new joy, new hope, and lenses of greater penetrating power, with which to get ever grander and more glorious comprehension of the grand and staggering wealth which eventually is to become man's.

We are of the column—our meetings are as milestones or stations, where each brings for the others' contemplation, study, pleasure, and enrichment, such trophies as he has been able to

acquire, on his arduous journey—for the journey is arduous; and the trophies, as time has shown, come only to him who is working on an up grade.

Other columns there are, the members of which are advancing with like hopes and like thrills, at all angles. Between and mingling with the ranks are the great throngs of unorganized workers, whose part in the voyage of life is the support of those in rank.

The other columns and the supporting workers constitute our public. Our relation to the public is of paramount importance. Could we render it a service 100% perfect we should cease to be; and, to the honor of our profession be it said, there is no member of our rank but is toiling to the best of his ability to this end.

Our standing as *men* must be measured by our relation with our fellows in the ranks, and our relation and service to the public—our fellow men. Honorable success is commensurate with honorable service. Trophies found, or if you will, dug out, and offered freely to our Fellows, advance us in the rank; as does excellency of technique in giving of the same to the public.

To the man who has, self forgetful, so conducted himself, all honor. Any amount of this world's goods that he may acquire are rightfully his, and no true man will envy him his wealth or honor.

In thinking along these lines I am reminded of a story. A man, emerging from a wooded field, saw a drove of hogs running excitedly now this way, now that. He thought they must be possessed of the Evil Spirit, until, seeing a man standing near a tree, he approached and asked what was the explanation. The man motioned him to come nearer, and in a whispered voice replied: "They are my hogs—before I lost my voice I used to come out and call them to their feed—then, having lost my voice, I trained them to come to the sound of my beating with a stick on this beech tree. It worked perfectly until lately, when the woodpeckers have reappeared. You have seen the result."

Of late years many woodpeckers have appeared, who have attracted the public, and, like the hogs, the public has been deceived.

These woodpeckers have heads of different colors. The meanest, most atrocious of all is on those men who, knowing the honest call, and not able to make it, have come out of our own

ranks, or who, wishing to remain in the ranks, hides his pecker-ing head, and thinks to escape discovery. They are our quacks and charlatans—they deceive the public, and commercialize the ignorance, love, and suffering of humanity.

In years gone by it was Mesmerism, or an Indian found herb or root, whose secret place of growth, healing power, etc., had been revealed to his missionary father, that the son was willing to bring to your service for cash.

Today it is the same to the nth degree—occultism, govern-ment of the subconscious mind, controlling the weal or woe of the body, or the little understood but powerful forces of Nature are advertised as mastered, harnessed, and at your service for cash.

Electricity, the X-ray, Radium and the like, used in some un-usual way, cures deafness, blindness, tuberculosis, cancer, etc.; but never when administered by an honest, self-respecting and upstanding man, such as belongs to this and like organizations, whose first thought, should he be able to find any such potently beneficent drug or power, would be to bring it, with all explana-tions, to his fellow members, that the benefits, multiplied by thousands of times, should come to suffering humanity.

This attitude of our profession is but little understood by the public. The public should be in some way educated, that they may know the tests by which to differentiate between the quack or charlatan and the true physician.

During the few years last past we have heard much in the Presidential addresses of this, and some of our sister societies, as to what constitutes the proper education of a specialist. This important matter has been pretty thoroughly thrashed out, and I think practically agreed upon. I should like now to propound, and to a limited degree, discuss, another question quite as per-tinent to us, if not more so, than the education of the specialist, to wit: What shall we do with our demobilized specialists?

By demobilized specialists I mean those men, who during the most active period of their lives, have had hospital and clinical connections, which have ceased to exist because of age limit.

Most great cities have many such from our own, and other specialties.

Probably the greatest asset of the medical profession is expe-rience, and judgment founded upon experience. This body of demobilized physicians and surgeons contains many who are in

the best of bodily and mental vigor—whose minds are rich storehouses of wisdom and good judgment, acquired from personal contact with the same problems which confront, and sometimes confound us.

To entirely disengage these men from their accustomed constructive work is to encourage the greatest wastage of acquired wealth.

Since being in Washington I have made inquiry as to what is the plan of the Government as to retiring its scientific men. A friend of mine who is the chief of one of the scientific bureaus in Washington tells me that the Civil Service Retirement Law calls for the retirement of all scientific employees at the age of seventy; but there is a provision that if the secretary of the particular department to which that employee belongs considers that the services of the man who has reached seventy are of value to the Government, and that he is physically capable of performing them, he may send a certificate to the Civil Service Commission, and request an additional appointment for a term of two years. At the expiration of this two years, the same thing may be repeated under the same conditions.

The bureau chief who was telling me this said that only the day before he had written a request to the secretary of his department asking that a certificate be issued for two more years to a scientist who is already seventy-six years of age, stating that he found, that, although the man could no longer make field trips, he was of enormous value at his desk on account of his great knowledge, and on account of the great influence he exercises over the younger men, through advice and kindly assistance of many sorts. And the new President of one of the great universities, in talking to a friend of mine the other night, said that one of the serious problems that had occurred to him was the keeping of certain retired professors—men of great culture and experience—on the campus and continually interested in the work of the university. He said that they created an atmosphere which could be gained in no other way; that they could be continually called upon for valuable advice based upon years of experience, and the fact that they were living there and continually interested, makes an impression on the student body, and upon the younger members of the faculty of enormous value to the university as a centre of research and culture.

Of late years, too, we have heard considerable talk of "Group

Medicine"; and, whereas, I believe, the central thought in the minds of men who have tried to further the practice of Group Medicine has been the benefits accruing to the patient, I feel so far the commercial benefits accruing to the members of the group has not been far in the background, and if so, I offer no criticism.

There might be, however, a higher, more pleasing aim, and a vastly greater and more satisfactory reward, if, with commercialism absolutely ruled out, there could be arranged a proper system of what I will call Group Work.

The aim should be scientific advancement, and the consequent lasting service to humanity: the reward, that wealth of feeling that must be his, who, rising above self consideration, has wrought for his fellow man.

For many years we have come together and discussed many and various moot questions; and today we seem no nearer to agreement, nor yet to an understanding.

The above holds especially true when we come to consider the diseases of the parts in which we are especially interested, in relation to the diseases of other parts of the body, or to so-called general disease or constitutional vice.

For instance, there is the question of arthritis, the relation of the diseases of the upper air passages to the disorders of the gastro-intestinal tract; optic diseases in relation to diseases of nose and accessory sinuses, etc., etc.

Much of the trouble arises from our human frailties, from single and distorted vision—nothing is seen as well by one eye as by two—nothing is seen and understood by one man as well as if it be seen by several men, discussed and seen again. As comprehending lookers we may be astigmatic on account of previously held beliefs, or hopes for certain findings—the wish being father of the thought. For this reason we often take the word of our best friends with a variable amount of salt. I can assure you that I have listened with much scepticism to many papers read by dear friends, and I am sure the reciprocal holds true; for, but a few years ago, one of our most respected, and by me, much admired members, came to the Manhattan Eye, Ear, and Throat Hospital, and meeting me said: "Coffin, at our last meeting in Atlantic City you said and claimed things that I did and do not believe. What have you got to show to substantiate your claim?"

Now it has seemed to me that much of this uncertainty, much of this Missourian attitude of mind could be avoided by Group Study or work.

Take for example the question of optic neuritis as related to diseases of the nasal accessory sinuses. Some of our best men hold quite different views as to the cause of the nerve disease, and quite as divergent views as to the method of handling the same, to say nothing of the different views as to symptoms and diagnostic signs presenting in such a case.

What should be easier than that a group comprising several rhinologists, several oculists, and one or two neurologists, should go over such cases, discuss them, and go over them again, and yet again, if necessary, for a final agreement upon diagnosis: the same method to be followed out in the matter of treatment, and in reporting of results. Like groups should be working along the same lines in every city large enough to have the group.

What body of men so well prepared and fitted for the work, to whom it would be so convenient to at least partially constitute each of the groups as the demobilized men to whom reference has been made?

There should be in each of the larger cities a new institution for group study, diagnosis, treatment, and periodical publication of findings, results, etc.

I can think of no way better calculated to enhance and crystalize scientific thought as to things medical, and thus to bring about greater benefits to mankind. I have talked this over with some of the best men in New York, and so far the unanimous opinion has been that the suggestion is timely, and if carried out, must result in a wonderful achievement of the aforesaid aims.

While explaining the plan to one of our best oculists of middle age, he broke in with: "Splendid! What a glorious thing to look forward to, if one might hope to belong to one of the groups. It's something to work for."

This plan would go far to educate the public as to the altruistic side of the true physician, and bring forcibly to the light the high ideals of the Medical Profession; and further, such a project carried out will tend mightily to fix the world's gaze on America as one of the great centres for medical education.

One word in regard to the work in our specialty. Last year

one of our honored members and producers said in his presidential address before another society, "Laryngology", and he used the term in its broad application, "has exhausted itself. We have reached our limits".

I wish simply to say that Baron le Ray, Napoleon's surgeon, in his time made the same statement in regard to surgery; and I feel certain that if Mosher will keep his pace for a few years his work will belie his words.

Our Secretary has asked me to urgently urge our younger members to do more writing, and to more frequently offer reports of their work for the programs. One word as to our programs, and I am through. Why should we not have a program committee which, at each meeting should agree upon one or two subjects to be discussed at the following meeting, announcing the subjects, and as far as possible, the discussions one year in advance?

Thanking you, gentlemen, for the honor you have paid me in electing me President of this vigorous Society, I declare the meeting for 1922 open for the furtherance of the regular program.

SOME PHASES OF ESOPHAGEAL STENOSIS.

By RICHMOND McKINNEY, M. D., Memphis, Tenn.

Through the development of methods of direct examination of the lower respiratory tract, the laryngologist has broadened his field by turning his technical skill to the study of esophageal conditions with the aid of the esophagoscope. We claim justification for this theft from another province in the progress that we have made in the diagnosis and treatment of esophageal lesions.

The clinical report that I am to make might be termed a study in diagnosis, for I seek here only to bring out some diagnostic features, and to show how important esophagoscopy is in determining the nature of a stenosis in this region.

Some six years ago I presented a thesis for admission to this Society in which was discussed simple inflammatory stenosis of the esophagus. At that time I could find very little in the literature of this subject other than some pioneer contributions by Guisez, in France, and Jackson, in this country. Their experience agreed with mine, in that there might slowly develop a chronic stenosis of the esophagus from a spasm due to local irritation, which might result in a thickening of the esophageal walls and stenosis that could prove dangerous to life, and yet be non-malignant. Since publication of this essay I have had an opportunity to observe several additional cases, the most interesting one of which came under my observation the year prior to publication of the thesis mentioned. The diagnosis here is perhaps debatable, but the case is submitted under this classification.

CASE REPORT.

May 1, 1914, Mr. M., aged 58, came to me with a history of a fullness in the throat and difficulty in swallowing solid food, dating back some seven years. He had not been losing weight, having always been thin. Sometimes food was regurgitated several hours after eating. There was no pain at all.

Esophagoscopic examination showed an annular constriction, beginning at the lower portion of the upper third of the esophagus, and extending downward several inches. This constriction yielded to dilatation with olive-shaped metal bougies, and after this was repeated several times, Mr. M.'s condition

seemed to be normal, as there was no further difficulty in swallowing. He was cautioned as to the possibility of return of the condition, and advised to consult me should he again experience difficulty in swallowing.

I did not again see this man, he having drifted entirely from my professional horizon, until September 1, 1921, when I was asked to see him, he being under the care of a local physician at the Baptist Memorial Hospital, the expediency of doing a gastrostomy being then under consideration, and my advice requested.

I found Mr. M. bedridden, emaciated and very weak. He told me that since having been treated by me he had experienced complete relief for quite a while afterward, but difficulty in deglutition again developing, he began to receive treatment at various hands. Two physicians had diagnosed his condition as malignant, and he had received two courses of X-ray treatments. There had been no improvement whatsoever from these treatments, and for some time he had been able to take only fluids, and not much of these. He was losing weight rapidly, but there was no pain. Radiographs showed an annular stricture, apparently five or six inches in length, beginning about midway of the esophagus, and extending downward toward the cardiac end. There was considerable thickening of the involved portion of the esophagus, apparently fibrous in character. The radiographer at the hospital, without having previously heard my opinion, expressed himself as believing that the trouble was of a simple inflammatory nature. On esophagoscopy I found a characteristic constriction of the esophagus, as described, there being a perfectly smooth surface the entire length of the stricture, as determined by the use of a small-sized Bruenings' esophagoscope. Wassermann was negative.

Immediately I began the practice of bouginage with Jackson's linen bougies, introduced through the esophagoscope. After the first treatment of this kind, Mr. M. was enabled to swallow much more freely than formerly, and in ten days was able to leave the hospital, walking without difficulty, had gained in weight and strength, and returned to his occupation of farming in Arkansas. About once in two weeks he returned for bouginage, and had no difficulty in eating even such solids as beefsteak.

Spending most of his time in the malarious delta land of Arkansas, Mr. M. fell victim to a severe attack of malaria, a recrudescence of a chronic condition, accompanied by a pleurisy, and about the middle of February died. A few days previous to that he had been in my office, and that morning had eaten an enormous breakfast, without difficulty, and had gained five pounds in weight in two months.

Allow me now briefly to emphasize some of the important features of this case. A diagnosis of malignancy, which had been made on two occasions, was perfectly natural without esophagoscopy, and without bearing in mind the duration of the condition. Lacking an autopsy, it is true that we cannot absolutely exclude carcinoma, but I have never seen an esophageal carcinoma lasting fourteen years, have never seen one entirely free from pain, and have never had a case that I could dilate and bring up to almost normal deglutition, as I did in this case. The diagnosis of carcinoma was arrived at owing to the man's

age, and the fact that he had a stricture of the esophagus. Here the value of esophagoscopy was evidenced. The esophagus showed no ulceration, no budding from the walls, and no irregularity. Through the esophagoscope, and without anesthesia, dilatation of the stricture was comparatively easy, and the use of such methods of precision obviated a wholly unnecessary gastrostomy.

A case of more than passing interest is that of:

B. C. L., male, aged 58, who came to my service at the Memphis General Hospital, February 25, 1922, with the following history.

Two months ago he began experiencing difficulty in swallowing. At that time he weighed 150 pounds. The difficulty in deglutition rapidly grew more pronounced, and he lost appetite and was greatly weakened. Everything he ate was immediately regurgitated, even fluids being largely rejected. He suffered with chronic constipation, and at the time he came to the hospital, had not had a movement in five days. Diagnosis made in the receiving ward of the hospital was partial pyloric obstruction. Wassermann was four plus. Radiography showed a stricture of the lower third of the esophagus, annular in shape, with no apparent thickening of the walls. There was also comparatively little dilatation of the esophagus above the stricture. The opinion of the radiographer was that the stricture was benign, and spasmodic in origin.

Esophagoscopy was done by me on the morning of February 28th. The man was very emaciated, and so weak as almost to be moribund. A large-sized Bruenings' esophagoscope was passed down to the constriction with great ease, as there were no teeth to obstruct. A small amount of 10% cocain was used to anesthetize the gums and hypopharynx. The picture clearly seen through the esophagoscope was that of a typical, uncomplicated esophagismus. There was no ulceration, and apparently no thickening of the walls. It was easy to pass a size 30 Jackson linen bougie through the stricture into the stomach, and the man was so greatly emaciated that the end of the bougie could be felt through the wall of the stomach. The opinion expressed by me was that the esophageal stricture was a spasm, with a deeper and more serious cause. I could not conceive of such great emaciation and weakness from an ordinary, uncomplicated cardiospasm of two months duration.

Four days later, the man steadily growing weaker, he again was esophagoscoped by me, but nothing different seen from the previous examination. I very carefully swabbed the walls of the esophagus, but obtained not even one drop of blood. The next day, at eleven o'clock, he died. Just before he died, he vomited a quantity of coffee-ground material.

Autopsy by the pathologist of the University of Tennessee showed postmortem esophageal findings identical with those of the esophagoscopy, but the pyloric end of the stomach was quite another story. Here was found partial obstruction, a large amount of thickening around the pylorus, and large ulcers of the pylorus and of the gastric mucosa in the immediate vicinity. The condition was regarded as syphilitic, and the tissues studied in the laboratory bore out this finding. There was no tumor formation. The thickening around the pylorus was an inflammatory fibrosis, and miliary gummata were found scattered throughout. The pathologist was convinced that the esophageal stricture was functional, and secondary to the syphilitic process from below.

In the absence of a carefully worked out pathologic study of this case, it would be perfectly natural, in the presence of extreme emaciation, epigastric pain, an esophageal stricture, and a cancerous age, to make a diagnosis of cancer, and at the regular Monday evening conference of the staff of the Memphis General Hospital, before the report of the pathologist had been submitted, the trend of opinion expressed was to this effect, but both the gross and microscopic pathology negatived this. The esophagoscope had its value here in determining the character of the esophageal stenosis.

Now turn to another picture:

Mrs. S., aged 45, was seen May 22, 1919. About four years previously she began having symptoms like "heart-burn", accompanied by difficulty in swallowing. This difficulty in swallowing gradually grew worse, until it became constant. At this time she could swallow only fluids, and no acids. She had lost a great deal of weight, weighing 132½ pounds, whereas her normal weight was 175. A diagnosis of cancer of the esophagus had been made, but she was fluoroscoped ten months previous to the time that I saw her, and cardiospasm diagnosed by the radiographer. The obstruction was at the lower end of the esophagus, where she at times had intense pain. Under fluoroscopy the barium milk could be seen to slowly trickle through the constricted cardiac end of the esophagus.

With a local anesthetic Mrs. S. was esophagoscoped, and a stricture was seen, annular in shape, at the cardiac end of the esophagus, this apparently being two or three inches in length. No attempt was made at this time to go through the stricture.

Subsequently, at intervals of two or three weeks, the esophagus was dilated with Sippey's metal olives, some improvement following, and food being swallowed more satisfactorily, but in May of the following year, after the lapse of several months without her having been seen by me, Mrs. S. returned with the statement that she was again experiencing much difficulty in swallowing, was losing weight rapidly, and had great pain at the epigastrium.

Under ether a Jackson esophagoscope was passed clear through the stricture, and into the stomach. The next day Mrs. S. began to eat full meals, and continued to do so, and in a few weeks' time had gained twenty pounds. Nearly a year later she reported that she was doing well. Mrs. S. then removed to Oklahoma, and a few months ago, some difficulty in deglutition again returning, she consulted a surgeon in her locality, and I get the vague report that he said her trouble was reflex, from some uterine disease, did a vaginal hysterectomy, and Mrs. S. has had no difficulty in swallowing since. Veritably a neurosis!

A haphazard diagnosis of carcinoma perhaps was justified in this case, even more than in the one first related, for the symptoms were more indicative of carcinoma. Without the findings of the esophagoscope, and the relief brought by its use, this woman probably would have died of starvation from inanition.

Spasm of the esophagus is rather common, but cases of spasm with the symptoms evidenced in this instance are not usual.

Another type of esophageal stenosis is exemplified in this case:

A man, aged 26, came to me with a history of increasing difficulty in swallowing over a period of two years. He dated the beginning of his trouble from a tonsillectomy, but this of course was to be ascribed to the human weakness to blame something or somebody for their ills. Pieces of salad and dry food were swallowed with difficulty. There was no regurgitation of food, and so far no loss of weight. No pain whatsoever.

Esophagoscopy revealed nothing abnormal. The tube passed with constriction from only the regular anatomical narrowings. But it was different when barium milk was swallowed, and its passage observed with the fluoroscope. The barium was seen to fill a small pocket, about the size of a hazel-nut, in the esophageal wall. This was a pulsion diverticulum, and when filled exerted pressure, which caused the esophageal stenosis.

These four cases of varying nature have been selected from a not inconsiderable number of cases that I have esophagoscoped to determine the cause of stenosis, and are presented to show how much depends upon an accurate diagnosis in such cases. They also serve to emphasize how absolutely essential a carefully made esophagoscopy is in determining the true character of many of these strictures. In esophagoscopy we have annexed a new field of work, but to its development by laryngologists is due most of the progress made in the diagnosis and treatment of esophageal strictures, no matter how caused.

Before closing this clinical report, I should like to urge that all cases of esophageal stenosis, no matter of what character, be investigated through the esophagoscope, and with this accurate means of study and of treatment in our hands, there is very little excuse for the blind methods still so commonly practiced.

DISCUSSION.

Dr. S. Iglauer, Cincinnati, O., said that a very interesting question was raised. In their enthusiasm for esophagoscopy they were apt to overlook fluoroscopy and X-ray diagnosis. It is unwise to put down the esophagoscope before taking the X-ray. Fluoroscopy gives one the funtional physiology of the esophagus and most advances will come by that means. In a case of a simple inflammatory process running along 14 years, one must seek the cause. Syphilis was apparently ruled out in this case. The lymph glands surrounding the esophagus are often involved and with the frequency of tuberculosis in the chest it is not surprising if a periesophagitis arises from lymph gland involvement. Diverticulum is due to traction of adhesions in the outer coats of the esophagus. The anatomical side is difficult, but Dr. Mosher is working on that. One lesion mentioned was in the cardiac end of the stomach, and another as being caused by irritation from the uterus. If that were so then cardiospasm appears to be due to reflexes of remote

origin. Dr. Mosher feels it is mechanical, due to pressure of the liver on the subdiaphragmatic esophagus.

Dr. R. C. Lynch, New Orleans, La., said that he had not come in contact with these cases. He could not see how so few dilatations caused such a wonderful result in such a short time. In cases diagnosed globus hystericus in New Orleans, and further examined, only one in twenty-five was really that condition. There were two gummata, and two leutic ulcers. One patient had a large piece of crab shell in the lower end of the esophagus. One old doctor who had no teeth, had a mass of chicken, the size of a lemon removed, and after that had no symptoms of cardiospasm.

Dr. J. W. Murphy, Cincinnati, O., said that one point needed emphasis. In passing the bougie through the stricture, it is not necessary to use the largest bougie, nor to use force, but gradual dilatation procures better results. In lye strictures in children, if one can get the child to swallow the string, the bougie can be passed easily.

Dr. S. Yankauer, New York City, said that spasm is usually supposed to be of the lower end of the esophagus, and to be non-existent in the upper part. In a patient, a man of middle life, there was a complaint of increasing difficulty in swallowing. The X-ray showed the bismuth column coming to a sudden stop. A diagnosis of carcinoma was made and a gastrostomy was done to relieve the emaciation. No carcinoma was found, but it was impossible to pass a bougie, unless under anesthesia when it passed easily without encountering thickening or growth. Under esophagoscopy it was impossible to find the lumen of the esophagus, and a string passed down disappeared at the point of obstruction. Under general anesthesia the spasm relaxed. Later someone made a diagnosis of diverticulum below the cricoid, and the man was treated for this condition, but it was really a spasm of the upper portion of the esophagus.

Dr. R. C. Myles, New York City, said that three cases of stenosis of the esophagus had disturbed his summer vacation: one was an old sport who telephoned that he could not swallow. He was told to come up and it was found that he had the end of a lobster's claw in the esophagus, which was removed. A minister, who had swallowed a wish bone was the next case. The third was an opinionated, strong minded lady, who in spite of her doctor's opinion to the contrary, insisted she had swallowed a chicken bone. This proved to be the case and the lady was very triumphant at its removal.

Dr. McKinney (closing), said he went 50-50 with Dr. Iglauer, in regard to the X-ray. He never examined a case esophagoscopically without using fluoroscopy before so doing. In regard to the quick result in the first case reported, he had been equally surprised, but the treatment had been continued for some time after in order to keep the esophagus patent.

PRECAUTIONS IN USING LOCAL ANESTHESIA WITH THE REPORT OF A DEATH.

By CURTIS C. EVES, M. D., Philadelphia, Pa.

The study of the susceptible patient and the method of administration present the most interesting and complex problems in the use of local anesthetics. For the average individual the moderate dosage of a recognized local anesthesia has little danger. It should not be overlooked in the daily rush of our routine that even the mildest of these drugs are highly toxic and that there are more than a few individuals who possess an idiosyncrasy for them.

Inasmuch as practically all drugs used for local anesthesia produce almost identical toxic symptoms, novocain or procain the least toxic of those in general use, will be referred to unless otherwise mentioned.

After the consideration of the hypersusceptible patient the next most important question is the method of administration.

Susceptibility. Of all the drugs to which a patient may think he is susceptible, his statements in reference to cocain and allied drugs should be considered most reliable. Individuals who have been poisoned by cocain or procain are more susceptible to the second administration. If no history of having had the drug used before is obtained, susceptibility of the patient should always be tested by using a small well diluted dose previous to giving the full amount necessary for anesthesia. For example, when procain is to be injected for the removal of tonsils, it is my practice to inject about ½ c. c. of a ¼ of 1 per cent. solution at a point to be used later; after waiting five minutes, if no symptoms are produced, to proceed with the full amount.

Similar precautions should be taken before any injection or before applying large amounts of strong solutions of cocain to the mucous membrane.

Administration. The guiding fact should be first to keep well within the limits of safety in the amount of the drug used. The dose of procain or novocain as given by different authors varies widely. Roth (1) after quoting from twenty-one authors states: "From the above references no definite conclusions can be

drawn as to the exact toxicity of novocain in man. Under certain condition 0.02 gm. (approximately 1/3 of a gr.) has caused poisoning symptoms while as much as 2 gm. (approximately 15 grains) has been given without producing symptoms". He further states: "The untoward results that have been reported in the literature from the use of novocain in operations about the head and face might well be accounted for by the fact that absorption may be rapid". "Individual susceptibility is marked in both animals and man."

The dose of cocain has also wide limitations according to the different authorities. ½ to ¾ grain is usually considered a safe dose.

To keep well within the safety limit 2 to 3 grains of procain and ½ to ¾ grain of cocain can be used in nose and throat surgery.

In the susceptible patient much smaller doses may cause serious toxemia, while in the non-susceptible individual large doses are safe if guarded by measures to reduce the rapidity of absorption. This can best be accomplished by injecting or applying the drug in only small quantities at a time combined with adrenalin solution or allied drugs which cause vaso-constriction.

Allen (2) in his book on Local Anaesthesia states: "It is believed that cocain once fixed by the body-cells (in combinations with them) is not liberated from these combinations as cocain, but as constituent products, ecgonin, etc.; Glasenap believes he has isolated ecgonin from the urine. These derivatives of cocain are slightly anesthetic and slightly toxic, but much less so than cocain. As a result of the preceding statements it can be said that cocain exerts its full anesthetic or toxic action but once, and if exhausted locally there will be no constitutional reaction."

For injective anesthesia, the smallest amount of the least toxic drug which produces the desired result should be injected slowly, changing the position of the point of the needle after each few drops are injected. Thus avoiding the danger of inducing any great amount directly into the circulation should the needle at any time enter a vessel. Owing to the great vascularity of the tissue surrounding the tonsil, massive single doses injected in one place for anesthesia should be condemned; as should also large cotton tampons hypersaturated with strong

solution of cocain placed in the nasal cavity and left there for a long time. In the applications to the nose and throat precaution should be used to guard against the dropping or running of the solution to other parts.

One should be constantly on watch for the first toxic symptoms and since they often occur suddenly and when least expected should guard himself by the knowledge of how best to combat them.

The first toxic symptoms of cocain are usually those of excitation—talkativeness, laughing, nervous fear, etc. This stage of excitation may be absent, and I believe usually is, when procain or novocain has been used. Nausea, palor, sweating, pain in the head, faintness and collapse combined with rapid pulse and a feeling of constriction of the chest may be seen as early symptoms.

It is a mistake to attribute such symptoms to the nervousness of the patient. They may suddenly terminate in convulsions and death. In severe toxemia, convulsions simulate epilepsy. Beginning with the head and neck followed by general tremor, short jerky spasms involve the entire body, are accompanied with cyanosis, tightly clenched teeth with frothy mucous exuding from the mouth, rapid weak pulse, and rapid labored respirations—the pupils may or may not be dilated. The convulsion lasts for a few minutes but after subsiding for a short time may be followed by others and death.

Treatment. If the patient is not already in the reclining position he should be placed so at once. Should the state of collapse continue heart stimulants should be administered by hypodermic, and inhalations of amyl nitrate or ammonia given. For the convulsive movements, although not tried by the author, inhalations of ether would seem to be by far the best, artificial respiration and massage of the heart should be practiced.

Quoting from Allen (3): "For nervous excitement or convulsions H. C. Wood recommends chloroform by inhalations, but it would appear that ether should be better, particularly if it be proved that the central action is associated with the same vasoconstriction and anemia that takes place in its local field of action; inhalations of ether, due to the tremendous congestion which it produces in these parts, should prove of great value, besides stimulating vasomotors, heart and respiration and controlling the convulsions; nitrous oxid would be equally as val-

uable; amyl nitrate, while producing the same congesting effect would not exert the same controlling influence upon the convulsions.

Engstadt (4) says in speaking of cocain poisoning: "In the first few cases I was called on to treat, strychnin and morphin in combination were used with marked benefit. But, as cases kept multiplying, I found the action of these drugs too slow, and I decided that there must be something to counteract the poison more rapidly when life was in extreme danger. It was necessary to find a remedy that could be administered at any time and be instantaneous in its action. I soon found ether to be the required drug. This was administered as ordinarily given to produce surgical narcosis. Ether stimulates the vasomotor, is a tonic to the heart muscles, stimulates the action of the respiratory centers of the brain and of the pneumograstric nerve, and increases the pulmonary circulation in the first stages. While cocain inhibits the action of the heart, especially on the right side, it has also a marked inhibitory action on the respiratory centers of the brain. Death may occur from feeble respiratory movements of the so-called Cheyne-Stokes type or asphyxia."

"To me ether has proved extremely valuable. It has saved what seemed hopeless cases. It stimulates the heart and the respiratory system almost instantly. The pulse becomes fuller at once and of normal tension. The marked mental excitement is allayed as the poison rapidly disappears. The individual regains consciousness as soon as the effect of the small amount of ether has disappeared."

Allen (5) further advises the use of opium and its alkaloids. He believes however, it is best used as a prophylactic or preventative by the preliminary use before the injection of the anesthetic "when it does certainly permit the use of much larger doses of the anesthetic and in this way acts as a direct antagonist."

Allen (6) does not think the injections of adrenalin advisable after the toxic symptoms. He believes they are caused by paralysis of the higher nerve centers, adrenalin exerting the same action upon these centers as it does locally, causes a prolonged retention of the toxins in the brain.

On the other hand Eggleston and Hatcher (7) and Braun (8), advise the infusion of epinephrin solution upon the recognition

of the first toxic symptoms to stimulate the action of the heart so that the toxic agent may be brought in contact with the liver where Eggleston and Hatcher believe the toxins are destroyed, quoting from Eggleston and Hatcher (7): "Since acute poisoning in man occurs where least expected, those who frequently employ local anesthetics should always be prepared to apply the three resuscitative measures of intervenous infusion of epinephrin, artificial respiration and cardiac massage. All three should be used in combination, since no one is efficient, except in rare instances." They base their belief upon extensive experiments upon cats—they were able to resuscitate cats after a rapid intervenous injection of doses of local anesthetics up to twice the usual fatal dose by using the above measures.

All recent articles written upon this subject claim that these drugs kill by paralysis of the heart and respiratory centers.

The Committee appointed by the Otolaryngological Section of A. M. A. to study the "Advantages and Disadvantages of Various Local Anesthetics in Nose and Throat Work" (8), after two years study and communication with the members of the Otolaryngological section report forty-seven deaths from local anesthesia, all except eight cases were tonsil cases. Of the cocain deaths it is appalling to note that one-fourth were due to "mistakes of druggist or nurse".

After carefully selecting the tonsil cases which had to contributing causes such as tuberculosis, heart disease, mistakes, etc., reported, I found nine deaths caused from injections of cocain ranging in strength from 0.2 per cent. to 3 per cent.

Twelve deaths from injections of procain solutions ranging in strength from 0.5 per cent. to 2 per cent.

In thirteen of these epinephrin or adrenalin was used in the solution.

In the procain cases eight had cocain applied to the pharyngical surface before injection.

Four had only procain injections.

The greater number of procain deaths in these selected cases occurred because of better reports given of them. In the full number of forty-seven, twenty-five were cocain deaths, fourteen were procain deaths. The other deaths were: one, from the use of alypin which is very toxic, and two from apothesin. Morphin injections had been given to approximately half of the cases. Procain or cocain was the only constant medication.

Becker (10) reports a death of a girl twenty-five years old, following an injection of about 100 mimims of 1 per cent. novocain for the removal of tonsils. The patient died suddenly with symptoms simulating an epileptic convulsion. A larger amount of solution from the same container had been used upon a sister in the same manner without any toxic symptoms.

Simpson (11) mentions a death of a male patient before a tonsil operation had been begun. Hypodermic morphin gr. 1/6 was given one-half hour before, followed by local application of 10 per cent. cocain solution fifteen minutes before. While in the act of injecting ½ of 1 per cent. novocain the patient was suddenly attacked with what Dr. Simpson thought was an epileptic seizure, followed in a short succession by six or eight more convulsions and died shortly after.

White (12) reports a tonsil case which was being prepared for operation in his clinic. "The patient, a woman aged 23, rather heavy set, was prepared for local tonsillectomy without the previous use of morphin as follows: The tonsils, pillars, and pharynx were swabbed with 20 per cent. solution of cocain with 10-15 drops of 1-1000 adrenalin to the ounce. This was followed in about ten minutes by the injection of a 1 per cent. solution of novocain. About three drachms of the solution was drawn up into the ordinary type of syringe used for this purpose, and the injection was made in the usual sites on the right side, and about the same amount was injected on the left side. About fifteen seconds after the last injection, the patient, with an exclamation, reeled forward in convulsions. Cyanosis was marked. The patient was laid flat on her back on the floor, the sphincter ani dilated, and artificial respiration begun, but the convulsions persisted and cyanosis was still very marked; the pupils were semi-dilated, the pulse very faint, and the respirations shallow. The heart was massaged, ether and adrenalin given subcutaneously, and the use of pulmotor was instituted. The convulsions ceased, no respiration was evident and there was no perceptible heart beat nor pulse. The artificial respiration was continued for about an hour, at the end of which time the patient was pronounced dead. There was no apparent cause for the condition. At the post-mortem all the organs were found normal, excepting the thymus which weighed about 50 grammes.

Diagnosis: "Status lymphaticus."

Case Report.

After operating upon nearly one thousand tonsil cases under local anesthesia without any serious symptoms—a patient I was carefully preparing for operation before a public clinic suddenly died before the operation had been begun. On March 15, 1921, R. A., a young man, eighteen years old, blond, of medium build, with rather effeminate features, was prepared for operation as follows: morphin sulp. gr. ¼ with atrophin sulp gr. 1/150 had been given twenty minutes previous. Ten per cent. solution of cocain was applied to the superior and inferior pole of each tonsil by placing a cotton-tipped applicator saturated with the solution for a few seconds, in each place.

Following this about 1½ drachms of a 1 per cent. procain solution containing 1 drop of adrenalin, solution 1/1000 to the drachm, (approximately 1⅕ grains of procain plus 2 drops of adrenalin solution), was injected outside of the capsule on each side. In about one minute after the last injection, while waiting for the anesthesia to take an effect the patient said he felt badly and looked very pale. His head was placed between his knees for perhaps another minute, when he began with a slight convulsive movement involving head and upper part of the body. He was at once placed upon a stretcher, by which time the convulsive movements involved the whole body. Short, rapid, jerky spasm lifting the extended legs and head and shoulders from the stretcher, was accompanied by a general tremor, marked cyanosis, eyes open, tightly clenched teeth, with biting of tongue and frothy mucus exuding from the mouth. The pulse was weak and rapid, becoming almost imperceptible.

The respirations were shallow and labored. After lasting one or two minutes the convulsions subsided to a general tremor with improvement of the pulse.

A second convulsion followed after which the patient was taken to the ward, where the pulmotor and other measures were used to resuscitate him without avail. He died in about thirty minutes.

Solution from the same bottle had been used for a patient just previous to this one without toxic symptoms.

Having been told before the clinic began that one of the patients to be operated upon was subject to epilepsy, I at first mistook the symptoms for an epileptic seizure.

The autopsy showed a much enlarged spleen and general hypertrophy of the lymphoid tissue throughout the body. The post-mortem diagnosis was "Status Lymphaticus." The fact remains that the patient died with symptoms of novocain poisoning.

To almost every laryngologist there comes a period of enthusiasm for the use of local anesthesia in tonsillectomy. To operate upon a conscious patient without pain or bleeding and to have a grateful patient, grateful especially because of the elimination of the fear of ether and its after effects is encouraging—add to this a more or less lengthy series of such cases, anesthetized with a comparatively small amount of a solution of less strength than the general surgeon uses in almost limitless quantities.

The enthusiasm of the young laryngologist is apt to relax his vigilance and to cause him to select too wide a field for the use of local anesthesia.

It needs to be constantly borne in mind that ether, except in selected cases, is the safer anesthesia.

There is a large number of selected cases in which local anesthesia is the method of choice. This is especially true in operations upon the nasal cavity, in pulmonary, renal, hepatic disease and in the aged.

Local anesthesia is especially contra-indicated when operating upon the nose or throat of children, highly nervous or neurotic subjects, in epileptic and static lymphatic cases, and in some cardiac condition.

Summarizing the Precautions in Using Local Anesthesia.

1. A careful history and physical examination of the patient especially to detect susceptibility, neurosis, epilepsy, cardiac lesions, and the existence of a lymphoid diathesis.
2. Elimination of patient's susceptibility to the drug used.
3. The use of the smallest amount of the least toxic drug well diluted which will produce anesthesia.
4. The addition of adrenalin or epinephrin to all solutions to reduce the rapidity of absorption.
5. The solutions should always be administered slowly. When injections are given every care should be taken not to enter a vessel.
6. The reclining position is to be preferred, thus relieving all strain from the heart.
7. It should be the *infallible* rule of every surgeon to freshly mix his own solutions and to keep the stronger solutions for surface application in a different colored container.
8. The operator should be prepared at all times to administer the measures to combat toxic symptoms.

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DISCUSSION.

Dr. George M. Coates, Philadelphia, Pa., said that this timely paper of Dr. Eves emphasized the fact that one knows very little about the causes of toxicity of the agents used as anesthetics. One thinks the patients hypersusceptible, but how can that be proven. Trying a small amount to see if the patient reacts unfavorably is unsatisfactory and the only method is that of a careful physical examination before operating. That should be done in any case, whether for local or for general anesthesia. If one does not want to use ether, that does not mean the patients are safe for local anesthesia. Certain precautions can be taken, not only in tonsil operations. The majority of accidents are due to mistakes made in preparing the solution, on the part of the druggist, the nurse or the operator. The deaths known personally were due to the use of strong solution instead of the weak one. If different colored containers are used this would be avoided. One can start with wiping off the pillars of the fauces with the strong solution, then put that aside. The Special Committee on Local Anesthesia, of the A. M. A., recommends the previous injection of morphin sulphate to diminish the toxic action of cocain, procain and similar drugs. Personally, the speaker said, he liked to use mass injections back of the tonsils, of very weak solution, in large amounts, and making the injection very slowly. Young operators are in a hurry to push in as much solution as possible, because the patient gags, yet if the operator takes his time, and gives the injection slowly, and combines it with adrenalin, he will have no trouble. One can operate in these cases almost immediately. A safe anesthetic for the tonsils is normal saline solution, injected in mass, back of the capsule, in considerable quantity, and slowly injected. If followed quickly by operation the anesthesia is good. One tonsil can be enucleated and the anesthesia given again, as the effect wears off quickly. The other tonsil can then be removed.

Dr. H. I. Lillie, Rochester, Minn., said that the physician must be confident he can get good results, or the patient loses confidence. One can get results with the use of scopolamin morphin before doing the local anesthesia. The pressure method is used, with one-fifth of one per cent. cocain solution and one drop of adrenalin. The injection is made so that the solution goes between the aponeuroses and the capsule of the tonsil, and acts as a natural dissector giving very little chance for absorption of the drug to take place. The needle, a large caliber one, is kept moving, so that if the vessel is injured, it need not enter the blood stream. Syncope, palpitation and sweats, need not necessarily be signs of toxemia. Mattas, of New Orleans, reported a patient who expired in the operating room, because a table fell over, and before anything had been done. If anesthesia had already been given, death would have been assigned to that cause. Physical examination should be made on all cases before any operation. Patients with various conditions such as angina pectoris, have been operated on without accident. Packing has been discarded, and nerve blocking used, and it is felt that absorption is much less. One case had been reported of a fatality from antrum puncture. The autopsy did not show any particular reason, and whether this should be attributed to cocain or not is doubtful. In regard to laboratory work, the comparison of the lethal dose for frogs, with that for human beings, does not hold.

Dr. Charles S. Means, Columbus, O., said that this subject interested every man who had had one of these unfortunate occurrences. He could relate one. Everybody has a different method and different anesthetic. The case in point was that of a nervous lady of 35 years, who suffered follicular tonsillitis every few weeks or months, and was in very ill health. For four years the speaker refused to operate, but finally she had such a bad attack, it was decided to remove the tonsils under local anesthesia. The family physician warned him that she fainted easily and this was expected. A 4 per cent. solution of cocain was used with 1/8 and 1/160 of morphine and strychnin hypodermically. After a second painting of cocain, an injection of 1/2 per cent. apophysin (Parke Davis) was used, 2 drachms being put back of each tonsil at 3 different places. The operation was never reached. The patient fell over dead. It was supposed at first that she had fainted. A blunt gold needle was used and no perforation occurred. The speaker said he could not believe the occurrence was due to the anesthetic. No post mortem was allowed. He thought an injection of water would have killed the patient.

Dr. R. B. Canfield, Ann Arbor, Mich, said that amyl nitrate had been mentioned as a drug suitable in this emergency. In experimental work it had been shown that in cocain death the heart always stops in diastole. This fact had not been mentioned. He had yet to see an autopsy performed in which it was shown that the heart had stopped in diastole in one of these cases. Therefore he maintained these deaths are not anesthetic deaths. He preferred benzoate of soda as an anesthetic. Amyl nitrate is dangerous. If ether is used it should be given hypodermically. He did not advise using local applications of a strong solution and then giving an injection. This would give the patient more than a maximal dose of local anesthetic. Cocain had come into bad odor because of many deaths, and various anesthetics had been used to replace it. Really the surgeon is the cause of the death. In 20 deaths, 2 followed cocain, and 18 novocain. The speaker said he had yet to see a death really caused by the local anesthetic. It was the administration of the drug. When swabbing the base of the tongue the patient swallows some of the anesthetic and this is followed by absorption. Often this is the cause of death after the administration of the drug.

Dr. Linn Emerson, Orange, N. J. said the fact had been impressed on his mind that many of these are adrenalin deaths. Men used to shoot in 30 minims of ordinary adrenalin. The speaker said that his associate refuses to take out tonsils, unless under general anesthesia, and they had lost a lot of patients that way. Finally he, himself, had to be the goat and agreed to give local anesthesia. Too much adrenalin and too much cocain are frequently used. He made up his own solution: one ounce of water to which are added 10 drops of 10 per cent. solution and three drops of adrenalin. On the average not more than half of this preparation was used. He said he got just as good anesthesia and just as good ischemia. He was accustomed to paint the pillars with a ten per cent. solution before injection. This method was satisfactory and after using ether anesthesia he went back to cocain. On reading the statistics of everybody who has had deaths, it would be found that they had used from two to twenty-five times as much cocain and adrenalin as was necessary. At the time of painting the tonsil pillars, it was his custom to have the nurse give one fourth morphin and one-hundred-fiftieth atropin, so that the patient received a combined morphin and atropin hypodermic 2 minutes before cocain injection, and operation was immediately performed.

Dr. Sonnenschein, Chicago, Ill., said that in regard to the use of ether as being preferred to local anesthesia in status lymphaticus, he thought thymus cases are as dangerous with ether as with local anesthesia. He had had the honor to serve on the committee on local anesthesia of the A. M. A. and in his experience he had found that many men were backward in telling about deaths because they fear to be drawn into medico-

legal complications with the family of the deceased. Indirect methods had to be found to get the facts. It was only, however, when cases are frankly reported with the courage and fearlessness shown by Dr. Eves, that the truth would be known. If more men would report cases, it could be done confidentially, without fear of legal complications, and much could be learned. Freudenthal showed ten years ago that one could have adrenalin deaths and that the caution suggested by the previous speaker was well taken.

Dr. B. N. Colver, Battle Creek, Mich. said that in his clinic local anesthesia was used from choice, but the technic had been varied from time to time. At the present time a one per cent. novocain solution was used in half an ounce of water before operation. The whole preparation was rarely used. Five minims of adrenalin were used. The preliminary trial dose should be used, as it also had its psychological effect on the patient. He felt that some of the symptoms and fatalities might be due to some reflex, rather than to the direct action of the drug. Every operation had its psychological swing. The operator would have the same feeling, if in the position of the patient, and it was well to feel that the patient was coming back to his normal psychological balance before beginning operation. This was gained by giving the anesthetic a little more time to work. When making up the solutions, it was a safeguard to color the strong solution with a little methylene blue, so as to avoid confusion, which was frequently a cause of death.

Dr. Harold Hays, New York City, said this subject was a necessary one. The number of deaths from local anesthesia were few, compared with the number of cases. Were the men willing to give general anesthesia with the chance of lung abscess, instead of local anesthesia. There are some precautions to mitigate the fear of the patient, such as the results of Gwathmey's work with synergistic analgesia, in which he uses magnesium sulphate with excellent psychological effect. The better plan is to do operations in the hospital, not in the office, and not in the sitting posture, which adds to palpitation and nervousness. Since so many solutions were erroneously made, the speaker said he made up his own ,from powders made up to a certain strength. He was glad mention had been made of adrenalin. Patients frequently complain of severe headache from cerebral constriction. In conclusion, the operator must use every psychological means to quiet the patient.

Dr. L. K Guggenheim, St. Louis, Mo., said that application of cocain before injection is unnecessary and adds toxic absorption. Injection can be made at 2 points, just under the anterior pillar above and below, and one at the posterior pillar of the tonsil. Was it possible that these deaths came from embolism? R. Gordon, in writing upon deaths from antral puncture, concluded that they come from air emboli. Surgeons frequently use metal syringes, supposed to be full, but often only half full. If this is injected between the pillar and tonsil, or into the tonsil itself, it may well be that air embolism can explain the many fatalities, especially with injection of one half per cent. novocain.

Dr. Eves (closing), said that it was due to the death he had, that this paper was prepared. The number of deaths occurring is surprising, but they are not reported. The speaker said he felt sure there was an absolute cocain death, with identical symptoms in every case, and closely simulating epileptic convulsions. A strong solution of any toxic drug causes death. Adrenalin is one factor for preventing absorption of cocain. There are two direct causes of death: (1) rapid absorption of the drug; (2) hypersusceptibility of the patient. He thought air embolism could be ruled out. In his own cases he had always used a glass syringe. He had never seen any case reported in the literature as due to air embolism. A needle forced into a ruptured vessel in the antrum might cause embolism, but with careful injection could hardly cause death.

SOME REMARKS ON OTITIC MALIGNANCY; REPORT OF TWO CASES. HEMORRHAGE CONTROLLED WITH A WOODEN PLUG.

By J. CLARENCE KEELER, M. D., Philadelphia, Pa.

Otitic malignancy as observed by the otologist is relatively an infrequent occurrence, if we may judge by the limited accounts in the text-books on diseases of the ear. Many authors fail to mention the ailment, others, make but a casual reference. This is probably due to the fact that the malignant growth is situated, though uncommonly, in that part of the external ear or temporal bone plainly visible.

When the "entire organ of hearing", the temporal bone and the adjacent soft tissues, the brain stem with its membranous coverings; the acoustic nerve, subject in a great degree, to neoplastic growths with sarcomatous and epithelial changes are considered, the otologist and surgeon, especially the intracranial surgeon have given fairly full explanations of the many stages in the development of malignancy.

Owing to the anatomical relationship of the internal ear to the cerebellum, the pons and the medulla-oblongata, the otitic surgeon is confronted, constantly, with lesions within the posterior fossa, which formerly were regarded as some obscure, incurable middle or internal ear disease, i. e., Meniere's or labyrinthine involvement; and many cases have a history of otorrhea with the usual associated symptoms that are not suggestive of being a neoplasm or malignant.

The genesis of intracranial involvement is usually indicated by an inequality of the face; a lack of sensitiveness to the skin surface of the affected side; a persistent and uncontrollable tinnitus; headache; impaired hearing; dizziness and inco-ordinate movements. At this stage, a Barany examination and a radiograph of the petrous bone are diagnostic helps.

In former years, an invasion of the subtentorial area by the otologic surgeon was regarded with disfavor; but since he has become more familiar with this surgical area, an earlier and a more sharply defined diagnosis may be made.

A neoplasm involving the internal auditory pathway, frequently originates in the acoustic nerve within the internal auditory canal or meatus, as a neuroma or neurofibroma, and has a marked tendency to undergo malignant change. It is usually of slow growth. Those of rapid growth do not differ in symptomatology, but there is a more sudden intensification of the existing symptoms.

Agreeable to the title of this paper, I shall endeavor to limit my remarks to the malignant phase only, the diagnosis having been confirmed by autopsy and the type of growth determined by microscopic examination.

The frequency of primary otitic malignancy may be estimated by the reports of otologists and their associates in the great Clinics. Permit me, therefore, to cite some instances.

(1) Bürkner collaborating with sixteen aural specialists, reporting 43,730 patients, found .32 in each one-thousand cases to be malignant tumors, (carcinoma and sarcoma).

(2) Rasmussen and Schmiegelow. Malignant tumors of the middle ear and auditory organ, generally belong to the comparatively great curiosities.

(3) Story. Primary malignant tumors of the tympanum or temporal bone are rare. Not a single case was observed during my seventeen years' work, in the largest hospital in Ireland where 1000 new cases were seen annually.

(4) Dalby. With twenty years' experience in private practice and St. Georges Hospital, London, but six cases were found. One was fatal owing to hemorrhage from erosion, into the internal carotid artery; the patient dying from suffocation while asleep; no record is available of the final results of the remaining five.

(5) Lee and Stockdale. Eye and Ear Infirmary, Liverpool. Only two cases of primary epithelioma of the auricle in our ten years' practice.

(6) Bezold. In 20,000 cases, carcinoma was found but four times.

(7) Schlitter. Among 40,000 diseased ears, only two cases of primary cancer of the middle ear were found, during the last twenty years.

Primary carcinoma of the middle ear without history of suppuration is quite unusual, though I believe that if a systematic histological examination were made of the tissue from the

tympanum before metastatic involvement occurred the statistics of its frequency might be considerably changed.

Primary malignancy of the auricle, beginning as a pimple or a wart occurs during the physiological decline of the patient. Politzer writes of a "spindle-celled sarcoma" which had completely filled the internal auditory canal. He also finds frost-bite a predisposing cause of angioma and in many cases there is a history of trauma. Instances cited:

(1) Trunecek, of Prague. Two cases, where injury to the auricle terminated in ulcer of the auditory meatus which rapidly increased in growth and bled freely.

(2) Denker of Copenhagen, removed a wart from an auricle. This was followed by ulceration and a discharge from the meatus, necessitating complete removal of the auricle and the cartilaginous canal. Skin-flaps were transplanted and later, skin-grafts by the Thiersch method, preserved the auditory meatus.

(3) Welkin reports a case of squamous-celled epithelioma treated with 1/500 and later by 1/300 solution of pyoktannin, by injection, greatly relieving the pain. There were no constitutional ill effects; the disease was temporarily checked, but the patient succumbed.

(4) Toynbee reports five cases of primary carcinoma in the middle ear, and believes it originated in the mucous membrane of the tympanic cavity.

(5) Sutton had 17 cases of primary carcinoma of the auricle, in 17 years' experience, all occurring in men ranging in age from 28 to 81 years.

(6) Schwartze reports 6 cases of primary carcinoma, all patients over the age of 50 years.

(7) Billroth saw a primary carcinoma which began in an apparently sound ear, in a woman 56 years old.

(8) Roosa and Schwartze stress the belief that chronic suppuration is the cause of malignancy. As cancer in other parts of the body is thought to be caused by chronic irritation, it would seem that a constant irritant, the result of necrosis, might develop malignant disease in an ear.

(9) Newhart. A most common factor in the development of cancer is the chemical irritation of a purulent discharge, upon the epithelial tissue.

(10) In 1862 Virchow, described a neuroma of the nervus acusticus which had undergone sarcomatous degeneration.

An early microscopic examination is indispensable for diagnosis in the primary stage. Hemorrhage and facial paralysis are prominent among the first symptoms. In unoperated cases, the prognosis is hopeless, unless there is early surgical intervention associated with Roentgen-rays and radium.

Summary: The literature investigated in this series includes 60 cases. The earliest case reported was in 1854 by Dr. Toynbee of London. There were 30 males and 22 females, in 8 cases sex was not mentioned. There were reported 30 cases of facial paralysis as one of the manifestations. The youngest case reported with sarcoma was a child, age 3 years. This form of malignancy is exceptionally rapid and fatal in childhood. Epithelioma of the middle and internal ear was found in a case 60 years old; and with the disease limited to the auricle, one case age 84 years was the age limit reported, denoting physiological decay favors the development of epithelial growth.

There were 30 cases of chronic otorrhea extending from 6 months to 63 years. The character of the discharge was always extremely fetid, and usually blood-stained. Meningitis occurring months after a mastoidectomy, suggests malignancy, especially if good drainage had been established.

The two cases following aptly illustrate the various phases of otitic malignancy of both the middle and internal ear by metastasis. First case from the parotid gland to the middle ear, and in the intracranial case, the premonitory symptoms were manifested by involvement of the facial, acoustic and the vestibular nerves, giving the first indications of the disease, though entirely unsuspected.

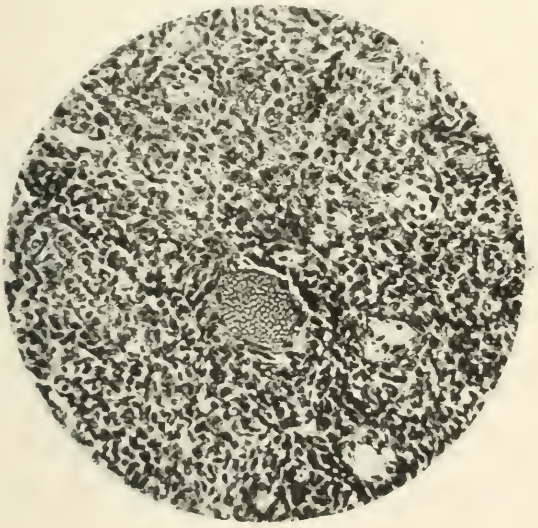
CASE REPORTS.

Jefferson Hospital, Philadelphia, Sept. 8, 1919.

Case 1. Male, age 48, German. Applied for relief of severe pain in the parotid gland, and on the right side of the head and face. Foul-smelling, blood-stained discharge existed during the past six months. There is an irregular shaped mass extending to the external auditory meatus. This feels like fibrous tissue. There is constant tinnitus and distressing dizziness. The hearing in this ear is completely lost.

Blood, Wassermann, negative. September 17, 1919, a radical mastoid. The cortex is eburnated. Many cells contain pus; others, filled with a dark serosanguineous fluid. Cell structure is soft and hemorrhagic, except a small sclerosed portion through which the mastoid emissary vein passes. This bleeds profusely, and is controlled only by the constant pressure of the assistant's finger, until the operation is completed, all other methods having failed.

Both attic and antrum contain cholesteatoma. The promontory is no longer present, the area serving for the attachment of the polypus. All diseased tissue is removed and the wound is closed in the usual manner. The patient recovered rapidly and insisted upon leaving the hospital, on the seventh day. The tenth day following operation, he returned to the hospital with an alarming secondary hemorrhage from the operated ear. The wound was reopened, under ether anesthesia. The hemorrhage was from the mastoid emissary vein, the bleeding of which, in the previous operation, had been so difficult to control. The course of the vein is parallel with the lateral sinus in the bone separating the cerebellum and the mastoid cavity. As a last resort to check the hemorrhage, a piece of wood was plugged into the bone. Immediately, the bleeding stopped. During the operation 1000 cc normal salt solution was administered, intravenously. The wound closed by granulation and there was an uninterrupted recovery.



No. 13508 B. Hemangioendothelioma. Higher power, showing type of cell in more detail and small blood-vessels filled with blood cells.

Post-operative treatment. Dermatization of the mastoid cavity was slow, but complete. A portion of the lamina spiralis is readily seen and can be palpated by the aid of an applicator. A small growth from extension of the parotid through the anterior wall of the tympanum was removed and found to be malignant. He was given radium treatment, the detail of which is herewith appended.

Treatments started, December 24, 1919. Last treatment, April 11, 1922.

One treatment, radium in back of mouth. 50 mg. 3 hrs. Filter 1 millimeter lead. 1 cm. cotton.

One treatment, radium embedded in growth at angle of jaw on right side. 50 mg. 6 hrs. Filter 1 mm. silver.

Two treatments, radium placed inside of ear. 50 mg. 1 hr. Filter 1 mm. silver and wax.

Seventeen treatments, radium on face and neck around right ear. 100 mg. the usual amount used but 4 times 90 mg. and 1 time 140 mg. were used. Time of treatment varied from 2 to 3½ hrs. in a single position. Usually 9 or 10 positions were treated at a single exposure by moving the 100 mg. pack of radium every 2 hrs. for 18 or 20 hrs. Filter 1 mm. lead, 1 cm. wood.

Histology:—The tissue is composed largely of solid masses of rather large, oval cells possessing nuclei which stain faintly. The masses of cells are separated by trabeculae, and on one margin is a squamous epithelial surface which is inflamed and ulcerated. The cells in the nodules are arranged to form whorls in places. There are numerous small thin-walled blood-vessels and small channels.

No. 13278 A. Endothelioma, probably arising from dura. Low power showing the numerous psammona bodies in the cellular areas.

Diagnosis:—Infected and inflamed hemangioendothelioma.

Jefferson Hospital, Philadelphia, November 19, 1920.

Case 2. B. G., Female, age 16, American. Presented with facial paralysis, right side, 9 months' duration. Epiglottis is turned to the right. There are no objective symptoms of mastoiditis or purulent otitis. Some thickening of the superior posterior osseous wall is suggestive of hyperostosis. Hearing is practically destroyed. Constant tinnitus and occipital pain. Attacks of vertigo. Radiograph of the right mastoid shows clouding and partial obstruction, probably subacute or chronic mastoiditis. The lateral sinus is not clearly shown.

The mastoid operation revealed a mass of bleeding, broken-down cells. There is no necrotic or surgical exposure of the dura, facial nerve or sigmoid sinus. A vein, the size of a superior petrosal sinus, bleeding freely, is seen in the upper posterior triangle of the mastoid. A serosanguineous fluid is found in some of the lower cells.

Post-operative. There is an improvement in the patient's general condition and recovery appears certain. But four months later there was a sudden exacerbation of the previous symptoms, and an acute meningitis developed causing her death.

Report, head autopsy: Dura is slightly thickened in places and the superficial vessels over the vertex of the brain show considerable infection. There is marked pial-edema, but no definite exudate is observed. The under surface of the right half of the cerebellum is densely adherent to a mass in the posterior fossa. This mass was also adherent to the posterior surface of the petrous portion of the temporal bone. The under surface of the cerebellum shows evidence of pressure, but there is no necrosis of the tissue. The vessels at the base are likewise infected, but no definite exudate is observed. On section, the brain shows no gross lesion. The ventricles contain clear fluid but are not distended. The mass in the right posterior fossa is firm, nodular, and tensely adherent to the petrous portion of the temporal bone. It is tough in consistency and at its thickest portion, measures about 2 cm. It extends downward, slightly encroaching upon the foramen magnum.

On removing the mass, the posterior surface of the petrous portion of the temporal bone is found to be very rough, but there is no evidence of infiltration on the bone by the tumor. The right middle ear is continuous with the mastoid cavity, and contains a reddish, rather firm inflammatory tissue; but the internal ear appeared normal.

Gross diagnosis. Tumor involving dura, right posterior fossa of the base of the skull, with pressure on right lobe of the cerebellum.

Bacteriology. Inoculation from the right middle ear yielded a pure culture of the staphylococcus albus.

Histological diagnosis. Endothelioma of right posterior fossa of the skull, probably arising from the dura, contains many psammona bodies. The tumor is composed of irregular cells arranged concentrically, with small, round calcified bodies. In other areas, there is considerable fibrous tissue.

Note.—I herewith express my deep indebtedness to Drs. Willis F. Manges, for his excellent radiographs, B. L. Crawford for his valuable laboratory work and William S. Newcomet for the administration of radium.

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DISCUSSION.

Dr. Gordon B. New, Rochester, Minn., said that he had had a slide made to show his own experience with malignancy of the middle ear. These growths could be divided into two groups: (1) primary middle ear epithelioma; (2) Secondary growths of the middle ear, the larger group; these were often stimulated to squamous cell growth. The type of tumor comprise: (1) basal cell epithelioma; (2) mixed tumors which may change to sarcoma or epithelioma; (3) tumors of the naso-pharynx secondarily involving the ear, of lympho-sarcomatous type; (4) hypernephroma involving the canal. True sarcoma of the ear had never been encountered by the speaker. He said he had never seen malignant tumor which had been entirely cured although many cases had done well for several years. Surgery in these cases is a hopeless measure and radium is the best treatment known at the present time.

Dr. Edward B. Dench, New York City, said that his experience had been very limited in malignant disease of the ear. In one case of an infant under one year, the disease was supposed to be simple mastoid, but was really small cell sarcoma invading the naso pharynx. The carotid was tied to shrink the growth, but the child died six months later. The ligature of the carotid had rather stimulated the growth if anything. The speaker had seen several cases involving the floor of the external auditory meatus. In one case the ear was taken off, the auditory meatus dissected out, and the glands of the neck removed in order to be thoroughly radical. The man lived till over 80 years. In one case of melano-sarcoma of the auricle, the patient recovered after excision. In fibro-sarcoma a local operation was done and the patient made a complete recovery. In another case where radical operation was done, the patient died. In a spindle cell epithelioma the tragus was taken off and the floor of the canal dissected out; the X-ray was used to prevent recurrence and the patient made a complete recovery.

Dr. J. G. Dwyer, New York City, said he had seen three cases. Two were operated on without previous diagnosis. The growth in one mastoid had filled the cavity. It was an epithelioma. Another case was recognized as operation was being done. The patient died. The third case had polyp in the ear. That was fatal. The disease, Dr. Dwyer said, is a primary one of the middle ear and mastoid, and is common if you include epithelioma in this group.

Dr. Perry Goldsmith, Toronto, said that growths of the Eustachian tube are primary. In a case seen a few months previously there was no suggestion of growth. Later there was bulging of the nasopharynx which later involved the middle ear, mastoid and carotid region. The mastoid was opened up to relieve the pressure, and the finger could be passed into the naso pharynx.

Dr. J. F. Culp, Harrisburg, Pa., said that he had had two cases; both died. In one he had suggested taking off the ear, but the man told him he was too fond of operating. The man later died of cranial complications. In another case where the condition was recognized, an extensive operation was done by Dr. Harris and himself. The patient died 6 months later.

Dr. Keeler (closing) said that if routine histological examination of the structure of the middle ear were made during the suppurative stage, malignant changes might be revealed in their incipiency. The second case cited stresses the importance of postmortem; without knowledge of contributory cause, death from meningitis instead of the endothelioma would have been recorded.

In 1909, a case of endothelioma of the auricle beginning as a wart in a man, age 77 years, was successfully removed by amputation of the auricle. This case was not listed with the two cases reported because I did not examine the growth microscopically.

I hoped some one would comment upon the "control of hemorrhage of the mastoid emissary vein with a wooden plug." I know hemorrhage has been controlled by plugging with wood during surgery upon the long bones; but in this case, the mastoid emissary vein ran in an independent channel. In the examination of a large number of temporal bones, a separate canal for the emissary vein was found only in one instance.

THE INTIMATE LYMPHATICS OF THE TRACHEA*

By JEROME FRANK STRAUSS, M. D., Chicago, Ill.

In a recently published and noteworthy series of studies on experimental lobar pneumonia by Blake and Cecil of the Army Medical School, there is among other strongly verified conclusions the statement that such pneumonia can only be produced experimentally by the direct installation of the various types of pneumococcus into the trachea. They state that the typical lobar lesions cannot be brought on by spraying the nose and throat with virulent suspensions of pneumococci, but if the organisms are permitted access to the lower trachea, pneumonia results. The work was carried out on a large series of monkeys by injecting with sterile precautions broth suspensions through a fine needle inserted into the lumen of the trachea between the tracheal cartilages just below the larynx. The injections were made without producing coughing or gagging.

The principle involved in this work recalls a paper of Freudenthal's published several years ago, discussing the treatment of intrathoracic conditions by intratracheal injections, in which the author offers some convincing proof that substances injected into the trachea do not pass beyond its lower limits. The same writer in the discussion following the reading of Dr. T. W. Moore's article on intratracheal injections read before this Society in 1919, said: "It is important to remember when treating the bronchi with this method that substances injected do not go down much below the level of the bifurcation."

In this connection the experiments of Coakley may also be recalled, in which after the injection of India ink solutions into the trachea of rabbits it was found after short periods of time, varying from fifteen minutes to two hours, that little or no pigment could be found in the trachea, bronchi, or alveoli, having disappeared, the author states, by lymphatic absorption.

These deductions, i. e., that suitable bacterial injections into the trachea produce lobar pneumonia,—that such injections (in doses not large enough to produce choking) probably never reach beyond the beginning of the primary bronchi,—and thirdly that there is definite evidence of absorption in the

*Candidate's Thesis recommended for publication by the Council.

mucous membrane of the tracheal wall,—these deductions very naturally awaken a degree of curiosity regarding the intimate lymphatics of the trachea, gratification of which is sought in vain among modern reference works. There appears to be available but few texts describing the actual lymph vessels of the windpipe, in all instances volumes of Continental origin, and in none is the subject given more than a brief paragraph. In the divisions on the lymphatics in the larger systems of anatomy there is no mention made of the lymph vessels, text and illustrations are limited solely to a brief exposition of the tracheal lymphatic glands. The special works on the subject refer without exception to the difficulty of injecting the mucosa of the tube for determination of the distribution of the smaller vessels.

Periodical literature is devoid of any contributions to the subject with the possible exception of the work of Winternitz, Smith and Robinson on the role of the tracheal mucous membrane in the pathogenesis of pneumonia—and in this paper details are conspicuously absent.

Laryngologists seem to have neglected the matter entirely, and on account of its relation and importance to the ever increasing field of tracheo-bronchoscopy, I venture to bring some observations before you.

II

In the fifth century B. C., Hippocrates, referring to his discovery of a "white blood-stream" and of vessels which take up nourishment from the bowel, is probably the first writer in history to mention the lymphatic system. Within three hundred years of his time we read of observations by Aristole of peculiar vessels containing a colorless fluid to be found accompanying certain groups of veins and nerves. Later there is recorded similar teachings by Erasistratus, Herophilus, and Galen. This fragmentary knowledge was lost during the passage of medieval ages and it was not until the middle of the seventeenth century, when Asellius of Pavia discovered the chyle vessels in animals, that the subject stimulated early modern anatomists. Following this demonstration there was a rapid succession of discoveries. The chyle vessels were found in man; Pecquet and Van Horne dissected the thoracic duct and cysterna chyli; lymphatics in the breast were isolated; and lymphatic trunks were demonstrated individually in various parts of the body. The eighteenth century marks the beginning of our modern

conception, for at this time injection of the lymphatic trunks was introduced and it was shortly demonstrated that the chyle stream and peripheral lymphatics were parts of a single general system.

The early efforts with injections were coarse and unsatisfactory. Air, milk, and various colored solutions were put into the larger lymphatic trunks under pressure, the ramifying branches being followed for varying distances. Much of our knowledge regarding the gross lymphatics results from the injection of mercury, a method introduced by Anton Nuck, and used consistently by anatomists for a long period. The method was a difficult one however, and only successful in the hands of a few trained men. The technique for which Fohmann is largely responsible, namely block or parenchymatous injections, was welcomed by workers in the anatomical field; by merely plunging the syringe into blocks of tissue or into the parenchyma of organs many small vessels could be filled and traced. When, later, a pressure apparatus was substituted for the syringe larger fields could be injected and further topographic advances were made.

The method of Gerota, presented in 1896, created a new era in the development of lymphatic investigation. Gerota recommended that the tissues be injected through a fine glass canula under pressure with a mixture consisting of Berlin blue, turpentine, and ether. This technique, endorsed by Poirer, Most, and other authorities, permits the tracing of the injected matter over greatly enlarged fields down to the smallest capillaries, and the collecting trunks can be traced to the receiving lymph nodes.

It is only since the work of Von Recklinghausen who discovered that the lymph spaces and lymph vessels were lined with endothelial cells, that we possess a somewhat modern conception of their structure. Their embryologic origin has been in doubt until almost the last decade. Sabin has determined that the first lymphatic elements to appear in the developing ovum are budding processes from the endothelial lining of the veins, which by innumerable branching processes invade the body tissues down to the ultimate capillaries. It has been conclusively shown that the progress of the advancing capillary is by ameboid cellular outgrowths of the growing endothelial cells (E. R. Clark), and that actual growth is accomplished by cell division in the endothelial wall. The first endothelial bud

sprouts out from the anterior cardinal vein to form the anterior lymph sac. An increasing series of buds unite to form plexuses which develop into additional sacs—"lymph hearts"—and from these organs continually advancing capillaries spread into the developing fetal tissues.

Accounting for the peculiar characteristic anastomosis of lymph conducting elements, it is interesting to note that investigators observing these advancing vessels under the microscope have demonstrated that the growing lymphatic tip has the remarkable chemico-physical power of avoiding blood capillaries while having an attractive affinity for lymphatic vessels.

The lymph *glands* have two sources of origin according to embryologists: (1) A primary group formed from the embryonal lymph sacs by the bridging of numerous bands of connective tissue covered with endothelial cells. These nodes are found in chains lying along the course of the larger and earlier formed blood vessels. (2) A secondary group developing along the course of the larger lymph vessels by the formation of plexuses into sinuses and the subsequent bridging of the sinuses with lymph cords,—follicles forming in the resulting trabeculae. These glands are found in the developed body with no apparent relation to performed venous structures.

III

The difficulties encountered in the injection of the smaller lymph vessels of the trachea are several in number. The mucous membrane is firmly adherent to the subjacent cartilaginous rings which are imbedded in a strong dense fibro-elastic membrane, and in comparison to the mucous lining of the nose is remarkably thin. Great difficulty has been experienced in attempting the injections in consequence of this firm tense attachment. With injections carried out on the cadaver the principal objection has been the rapid destruction of the thin mucous surface due to evaporation and autodigestion. In the experimental injections made in living animals, after exposure of the trachea by open operation results have been unsatisfactory because of the escape of the injected fluid into the tissues and because the operative procedure introduces the foreign substance into artificially opened lymph spaces and vessels, producing innumerable artifacts and errors which render subsequent deductions questionable.

It has occurred to me recently that probably many of the difficulties might be overcome by endoscopal injections of a proper substance into the tracheal mucous membrane of living animals with examination of the resulting absorption at varying periods post mortem. This method would obviate the necessity of injuring the windpipe and peritracheal tissues, and without interfering with the normal physiologic conditions and functions of the organ, would permit investigation of the real network of origin of the lymphatics in its mucous membrane.

Dogs were chosen for most of the present experiments on account of the more favorable diameter of the lumen of the trachea, rabbits proving too small for satisfactory endoscopic work. Young dogs were found in preliminary investigations to lend themselves more readily to the work than adults, it being noticed in many instances that the mucous membrane was less firmly attached to the inner surfaces of the cartilaginous rings than in older animals. It was also remarked that though the normally thin membrane was if anything more thin in younger tracheas, they possessed, on the other hand, the valuable asset of greater elasticity, the membrane of adult animals proving very friable with small submucous injections. Thin dogs were considered to be most desirable as it was possible to control the position and movements of the trachea externally with the hand, and short necked animals were found to allow more exacting control during the actual injecting as they permitted shorter shanked needles to be used with consequent better leverage.

The following individual methods of technique were devised and carried out with varying degrees of success:

(1) The animals were anesthetized with ether after the administration hypodermatically of morphine, and the Bruning electroscope was passed into the trachea. Injections were made with a record syringe fitted with suitable sizes of the long shanked needles used at one time by Dr. Lynch and possibly still used by him for injections into the larynx. It was found to be essential that the needle point be rather short and of fine calibre in order to prevent laceration of the mucous membrane, and this danger was further guarded against by having the needle point bent into its shank at an angle of about fifteen degrees, thus allowing introduction of the point approximately parallel with the surface of the membrane. As a precaution against too great a pressure of fluid from the syringe the plunger was discarded and

a small rubber bulb was capped over the open end. In this way the injection was more readily controlled, the rate of flow being easily regulated by gentle pressure of the fingers on the bulb.

The first injected substance used consisted of a thoroughly agitated twenty-five per cent. solution of fresh India ink. It seemed advisable to dilute the solution, the stock ink being apparently too thick and oily to pass readily through the minute lymph vessels. Small quantities of the suspension varying from one-tenth to one-quarter of a minin were used after the first injections, larger amounts rupturing the mucous membrane with a spattering of the black fluid over the surface of the trachea. The ink injections proved to be of only partial value as was to be expected. The lymphatic network could only be determined in small areas immediately surrounding the point of injection and only after almost immediate autopsy. In autopsies made eighteen to twenty-four hours after injection the ink was found only at the point of needle entry. In some of these cases however we were able to demonstrate large collecting vessels outside of the trachea by reason of engorgement with carbon bearing lymphocytes; and in all the cases carbon deposits were found by microscopic examination in the glands of the recurrent chain and deep internal jugular nodes.

(2) The same technique was carried out and a far more satisfactory demonstration of the lymphatic network was obtained by substituting for the India ink an aqueous solution of Berlin blue. This stain, which has a special predilection for endothelical cells was made according to the following formula:

Berlin blue	1 part
Oxalic acid	1 part
Water, distilled	12 parts

In autopsies made within two hours after injection the lymph vessels show up as a fine blue radiating network, the details of which are visible to the naked eye, and very clearly defined with an ordinary reading lens.

(3) The experiment was repeated using a one-half of one per cent. solution of silver nitrate, after the method of Golgi. The method was discarded after a few trials as the medium permeated the tissues indiscriminately, and after exposure to sunlight stained the mucous membrane more like a contrast stain than a selective one.

(4) Intracheal injections of India ink were made through a needle plunged into the trachea between two of its upper rings

in a manner similar to the method used by Blake and Cecil for the introduction of pneumococci into the trachea. In these cases no discoloring of the lymphatic vessels could be determined at autopsy three hours later, but it was evident that some lymphatic absorption had occurred as carbon particles were found on microscopic examination of the peritracheal lymph nodes. Animals treated in this way were kept for a time under close post-operative observation, and in nearly all instances black streaks were apparent in the mucous secretion from the mouth after the first half hour. It is therefore probable that a very large factor in the disappearance of inert substances injected into the lumen of the trachea is not absorption but the normal physiological action of the ciliated epithelium of the tracheal mucous membrane.

(5) The above technique was repeated substituting Berlin blue for India ink. There was no macroscopic evidence of absorption and none could be determined under the microscope.

(6) In an effort made to secure a larger field of injection the endotracheal method described above (2) was repeated with modifications. After introduction of the electroscope a fine dry needle was inserted externally through the tissues of the neck into the tracheal lumen. After localizing the position of the point with the eye the needle was withdrawn from the lumen and, guided endoscopically, was reinstated into the lateral wall. This maneuvre permitted the use of a longer needle point, the axis being parallel with the interspace, and there was consequently less danger of back flow along the course of the needle track.

(7) In conjunction with the latter method, more through accident than by design, a large quantity of the blue stain was injected into the peritracheal tissues, and post-mortem examination was made in one case after one hour, and in a second at twenty-four hours. In the first instance there was a general flooding and staining of all the membranous structures, the dye having spread upward to a short distance from the occipital bone and downward to the bifurcation—guided by the fascial planes. The second case (after twenty-four hours) showed a blue tinted discoloration at the needle puncture, but the only evidence of flooding of the peritracheal spaces was a faintly green tinge in the membranes. The probable explanation of this phenomenon is that the stain, passing through the lymph spaces, entered the lymphatic vessels and attacking the nuclei

of the endothelium, caused a subsequent desquamation, the liberated cells carrying off the foreign matter.

In general the injections were made in groups of three at two levels; three in the upper trachea within two centimeters of the cricoid ring, and three just above the bifurcation. Each set consisted of one in the anterior wall, one in the lateral wall, and one posterior injection, the latter being made in the membranous strip between the open portion of the tracheal rings.

It must be stated in fairness to the reader that only a small proportion of the many injections made proved of value. In some instances no traces of the supposedly injected fluid could be found, due probably to an overzealous attempt to inject very minute quantities to avoid rupture of the vessels; and many times it was found post mortem (especially before a short needle was used) that the point had penetrated the tracheal wall and emptied its contents into the loose areolar tissue of the peritracheal spaces. In many cases the needle, however gently manipulated, tore through the delicate mucous membrane and the stain was spattered freely into the lumen of the trachea. Even in many of the successful experiments the limitations of the various methods used was apparent. The lymphatic course could be followed in most instances for only a relatively short distance from any one point of injection as far as the superficial network was concerned, though it was possible to illustrate the continuous routes of absorption in many of the experiments, connecting the tracheal efferents with their receiving nodes in the general lymphatic system.

IV

A study of the findings in a large number of tracheal injections indicates:

(1) That there are practically no lymph vessels other than the fine intercellular lymph spaces in the mucous membrane where it passes over and is attached to the cartilaginous rings.

(2) The tendency of lymph flow in this network of origin on the surface of the cartilages is upward and downward to the nearest interspaces.

(3) When the injected fluid reaches the interspaces, (or as most frequently occurred, was injected directly into the interspaces) the course of absorption becomes horizontal, running into small collecting vessels lying between the rings. These tiny trunks vary from one to three in number, usually single in the cervical portion and increasing at lower levels of the tube.

(4) The lower ring surfaces are somewhat more easily injected and the fluid shows a tendency to spread with equal ease to the next upper or lower interspace. Higher in the trachea there is a distinct tendency for the absorbed pigment to migrate upward to the first neighboring interspace rather than downward. It is also noted at the central portions of the tube that the superficial network is consistently less dense than at either the upper or lower levels.

(5) In the anterior wall injections the fluid crosses the midline with ease and shows an equal tendency to spread horizontally to both lateral walls, with a marked inclination to circumscribe the tube.

(6) The lateral injections, and this holds for both upper and lower levels, may spread anteriorly but the characteristic tendency is posteriorly to reach the membranous portion of the trachea.

(7) The posterior injections into the membranous wall demonstrate the presence of a large number of lymph vessels, and larger individual vessels at any given level compared to the number and size of the vascular elements in the cartilaginous portion of the tube. Here also is apparently a greater tendency to upward and downward communication, the incomplete cartilages offering no obstruction here. In the pars membrana, it may also be demonstrated, the lymphatic vessels are more numerous and of larger calibre in the thoracic portion compared to higher levels.

(8) The horizontal collecting vessels are more numerous in the lower interspaces and seem to increase in calibre in the interspaces near the bifurcation. They are joined in their course by minute collectors from the surface of the two adjacent rings, and the general direction of their flow is toward the posterior membranous wall. There is a disappearance of the majority of these trunks beyond the open ends of the cartilaginous rings in a radiating network of fine vessels which cover a comparatively large area in the membranous wall—a network which usually extends across the midline to similar vessels on the opposite side. It is here according to Poirer, Most, and few other investigators that the efferents leave the tracheal wall to join the general lymphatic system.

(9) Though there is a vast anastomosis of these vessels in the peritracheal connective tissue between the windpipe and the

esophagus, by far the greater portion of the main trunks join the lymph vessels accompanying the recurrent laryngeal nerve. These large collectors are interrupted in their course by nodes—the paratracheal glands—corresponding to the recurrent chain in man. From these nodes drainage to the deeper internal jugular groups can be traced by the deposits of the carbon particles of the India ink.

Conclusions

There is a partial lymphatic absorption of inert substances by the tracheal mucous membrane in a normal uninjured and uninflamed state.

The cartilage rings, dividing the superficial network of origin into segments, offer successive lines of defense against lymphatic spread of tracheal infection.

Absorption from the mucous membrane when no inflammatory condition exists, is less active in the middle third of the trachea than in either the subcricoid area or that portion approaching the bifurcation.

The tendency of lymph drainage at any given segment is to the posterior wall of the trachea, through which offending matter is carried from the field to neighboring lymph nodes.

Though the lymphatic function is segmented by the tracheal rings, and in this way may be considered regional, nevertheless anastomosis between the various levels is free by way of the plexuses in the posterior wall of the tube. There is also, to a very minor degree, an intersegmental drainage by way of individual capillaries which at irregular intervals hurdle the belly of the cartilages from interspace to interspace.

Hypothetically substituting pneumococci for carbon particles and stains, pneumonia may be caused by absorption of the organisms through the mucous membrane of the trachea into the recurrent nodes and jugular chain, arriving at the lungs by way of lymphatic ducts, great veins, right heart, and pulmonary artery.

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I. TWO CASES OF MASTOIDITIS, SINUS THROMBOSIS, FACIAL PARALYSIS ON OPPOSITE SIDE, DOUBLE CHOKED DISC: RECOVERY.

II. THREE CASES OF MENINGITIS DEVELOPING SIX WEEKS TO FOUR MONTHS AFTER MASTOIDECTOMY.

III. RUPTURE OF CAROTID ARTERY: RESULT OF CHRONIC MASTOIDITIS.

By WESLEY C. BOWERS, M. D., New York City.

CASE I.

History. J. P., aged 20, was admitted to Bellevue Hospital, New York City, on November 4th, 1921, complaining of pain in and discharge from the right ear. One week before admission she had a sore throat, pain in the right ear and some temperature. The sore throat cleared up in two days but the pain in the ear persisted and three days later—four days previous to admission—discharge appeared and had persisted. The pain had been slight in the day time but severe at night. The patient gave a history of having had trouble with this ear about five times previously, the first time when a very small child. Each time the condition cleared up in three or four days and there was no discharge from the ear between attacks. An incision was once made, when she was very young, behind this ear for an abscess.

Physical examination showed a well nourished Italian girl, apparently only moderately ill. With the exception of the ear condition she was normal, physically, in every way. Examination of the ear showed the meatus swollen and filled with purulent secretion. The swelling of the canal precluded a view of the drum. There was slight mastoid tenderness over the antrum and tip. A scar behind the ear about one inch long, extended from the tip of the mastoid upwards. This was apparently a Wilde incision. The temperature was 101°, pulse 104, respirations 20. Culture from the ear showed staphylococcus albus. The blood count was WBC 12,800; polys., 80%, lymph., 20%. X-ray of the right mastoid showed a slight degree of peri-auricular sclerosis; there was no evidence of bone necrosis; the mastoid was of the diploic type.

For the two days following her admission to the hospital, her temperature remained around 101°. On the third day it went suddenly to 105.3°. The swelling of the canal wall had lessened so that the drum could be seen. The canal was foreshortened and filled with thick pus. The eyegrounds were negative; the lungs clear; no signs of meningeal or cranial nerve involvement were present; the skin showed no petechia or rose-spots; there was no joint involvement.

Operation. The following day the mastoid was opened. It was found to be entirely broken down and filled with pus and granulations. There was a large abscess cavity over the sinus. The sinus was exposed for a distance of one inch. The upper part of the sinus wall was thickened; the lower part

obliterated; the wall was necrotic. Both ends were packed off, a free opening made in the wall and free bleeding obtained from both ends. The jugular was then tied off above the facial.

Clinical course. The night following the operation, the patient had a chill. In the course of the following three days the temperature gradually came down to normal. The patient appeared toxic, had some headache, was mentally obscured but not actively delirious. There was a slight positive Kernig. The blood culture taken before operation was negative. The spinal fluid showed normal pressure, was clear with four cells to the cm.; culture, negative. After the third day post-operation, the patient ran a slightly septic temperature between 98.4° and 102°, for six days following which it gradually come down to normal. On the 18th and 19th days, it was 97.2°; on the four succeeding days it ranged from 97.1° to 100°.

The following neurological note was made on the fourth day post-operation:

"Cranial nerves normal; no evidence of the cerebellar or cerebral abscess or pachymeningitis; the only evidence of central nervous involvement is a weak clonus on the right and left, which is compatible with the toxic, irritative symptoms."

On the seventh day after operation the patient's right elbow became painful, reddened and considerably swollen. This condition cleared up in about seven days. Blood cultures taken during this period were negative. On the eighth day post-operation, the blood culture was positive for staphylococcus albus. At this time she had four or five superficial abscesses on various parts of her body.

Fourteen days post-operation, the entire left side of her face (the right mastoid was the one involved) became partially paralyzed. There was a double choked disc; right 3 D, left 2 D. She had a spontaneous nystagmus in both directions, more marked to the left. She complained of some headache otherwise there were no meningeal signs. She appeared markedly toxic, very much prostrated, had numerous abscesses and complained of abdominal pain.

One neurologist was so certain of a cerebro-pontine-angle abscess on the left side that he advised exploration sixteen days post-operative.

Dr. Foster Kennedy's neurological examination, made five days after the first neurological examination and ten days after the development of the facial, was as follows:

"I am unable to decide about this case. The fundi have a high degree of exudate in lumps, outside the discs which are not completely obscured; an appearance more characteristic of toxic retinitis than of papillo-edema due to increased pressure. Kernig's sign is no more positive than is usual in mastoiditis with a local pachymeningitis. Nystagmus is present, coarser to the left than to the right. There is left facial weakness, quite incomplete, probably—though not certainly—peripheral. There is no fifth nerve palsy; neck not especially stiff; abdominal reflexes, left greater than right; other reflexes normal; clonus, negligible. No ataxia whatever. The likelihood of there being a frank pus focus at present, seems to me very small. The cerebellum is not functioning perfectly but not as imperfectly as it should if it were the seat of an abscess as large as one would judge if the fundi condition were to be considered as due to increased pressure."

The examination of the eyes, two days later by Dr. Wolff, follows:

"Both eyes show a marked edematous swelling of discs with elevation of about 4 D. Edema extends for a considerable distance beyond the disc into

the retina, so that the vessels shade off gradually to the lower level, instead of making a sharp bend around the swollen disc margins, as in the pure papillo-edema. The retinal vessels are very full and tortuous. On the surface of the disc, and extending beyond its borders, are numerous radially arranged, glistening exudates. There are a few hemorrhages visible."

Spinal fluid at this time showed normal pressure, clear, with 5 cells to the cm.; culture, negative. The blood culture was also negative. The blood count taken twenty-two days post-operation, gave WBC 15,600; polys., 66%, lymph., 33%, eos, 1%.

In the course of the next ten days, all symptoms disappeared, the facial paralysis entirely cleared up, the eye-grounds returned to normal. The patient was discharged from the hospital thirty-six days after operation entirely recovered in every way.

CASE II.

History. P. W., aged 19, was admitted to St. Luke's Hospital, New York City, on November 19th, 1921. Two weeks previous to admission the patient began to have severe pains running down the back of the neck and in the occipital region. These were constant and not relieved by anything. Two days later he began to feel feverish and acutely ill. One week before admission he started having chills—the temperature going to 106° on two occasions—lasting about half an hour and leaving him very exhausted. Pain appeared in the right ear, increased on movement of the body. A hacking cough developed and, on admission, a diagnosis of broncho-pneumonia was made. The patient stated that for some years he had had some pain and fullness, on and off in the right ear. Two years ago he developed an acute otitis media on the right side. At this time the drum was incised at St. Luke's and the otitis cleared up promptly. The canal had remained dry up to the present time.

Physical examination showed an acutely ill patient, with a hacking cough; temperature, 104°, pulse, 104, respirations, 22. Eyes: negative. Ears: considerable mastoid tenderness on the right side, canal dry, drum perforated in lower posterior quadrant; remainder of drum thickened. Left ear, negative. Nervous system: negative. Other examinations: negative. Blood count WBC 20,000, polys., 74%, lymph., 26%. Blood culture was negative. Spinal fluid clear, under normal pressure; four cells to the cm., lymph. 100%.

X-ray of the mastoids showed breaking down of the mastoid cells on the right side. The labyrinth re-acted normally to caloric and rotation. A moderate voice could be heard on the right side, one and a half feet (with the noise apparatus) and twenty-five feet on the left side. There was a Weber to the right. The upper limit was 22,802, right side; 29,802 on the left. The low limit was 220, right; 16, left.

These findings indicated an acute exacerbation of a chronic mastoid with the possibility of a sinus thrombosis. There was however, no history of discharge from the ear except for a short time following the acute attack of otitis media.

Operation. Operation disclosed a very large mastoid with the cells all necrotic and filled with granulations and pus. The sinus was far forward. There was a perisinus abscess. There was a large cholesteatoma in a cavity posterior to the antrum and in the middle ear. A large area of dura, in the middle

fossa, had been exposed by disease and was covered with granulations. The sinus wall was very much thickened and because of the history of chills with sudden rise of temperature it was deemed best to open the sinus. Both ends were packed off and a free opening made in the sinus wall. Free bleeding was obtained from both ends. The jugular was then tied off above the facial.

Clinical Course. The night following the operation the patient had two chills. The temperature came down to normal the following day and remained so. The pulse varied from 75 to 90.

As I was off service at this time, I did not see this case for twenty days after the operation. On the twentieth day post-operation, I again examined him. At this time his right pupil was larger than his left and there seemed to be some weakness of the right rectus muscle. There was no nystagmus; the fundi showed the disc O D, 3D, O S, 4 1/2D. The entire face on the left side (the right was the operated side) showed decided weakness. Questioning him, I learned that he had been unable to whistle for one week past (two weeks post-op.), he had had some headache off and on during this period, but not severe. The blood count at this time was WBC 9,600; polys., 63%, lymph., 37%. Spinal fluid was clear, four cells to the cm., lymph. 100%.

One week later, twenty-seven days after operation, the spinal fluid was negative for everything, including Wassermann. The eye-grounds showed O D, slight blurring; O S, 3D, and some retinal hemorrhage. The other symptoms remained the same and he had no headache. Six weeks post-operation there was no headache, the right pupil was still large, the left side of the face continued weak. The radical cavity had completely dermatized and was dry, with a small opening persisting behind, due to the fact that it was necessary to leave the incision open at the time of operation in order to remove the packing from the sinus.

Four months after operation, the facial paralysis had cleared up, the right pupil was still larger than the left, the fundi were clear. The boy felt and acted perfectly well.

Conclusions. It would seem that in these cases, the facial paralysis on the opposite side was purely toxic in origin. The "choked disc" may have been toxic or may have been a true choked disc due to the sinus thrombosis. The fact that the choked disc and the facial did not appear until two weeks after the operation on the jugular and the fact that the condition cleared up so completely would seem to indicate that the whole condition was toxic. The first case cleared up in about three weeks; the second in about three months.

I have been unable to find in the literature, any record of facial paralysis occurring on the opposite side, following mastoidectomy, excepting those due to basilar meningitis. I should be very glad to have the opinion of others as to the cause.

Three Cases of Meningitis Developing Six Weeks to Four Months After Mastoidectomy.

CASE I. (Six weeks).

History. C. L., aged 4, was admitted to Mountainside Hospital, Montclair, New Jersey, May 28th, 1919, with a temperature of 101.3°, pulse 110, respirations 24. She had a myringotomy performed on both ears, by myself, seven

days previous to admission. During the interim, her temperature had reached 104° several times.

Physical examination of the patient was negative except for the ear condition. Both ears were discharging profusely; both fundi were somewhat narrowed; there was moderate mastoid tenderness.

Operation. Operation showed both mastoids softened throughout. The left mastoid was more extensive than the right. An area of dura the size of a dime was exposed on either side. Culture from the wounds showed short-chained streptococcus.

Clinical Course. The child remained in the hospital twelve days. Following the operation, the temperature went to 104°, coming down to 99° in five days. It then went to 102° for two days, returning to 99° where it remained until she was discharged. The pulse ranged from 120 to 140. The mastoid wounds healed up and the hearing returned in about four weeks.

Six weeks post-operation and two weeks after the wounds were healed, the mother brought the child to me complaining that she would wake up about eleven o'clock every night and cry for about an hour, then go to sleep again. Otherwise, she seemed happy and well; played with the other children and had a good appetite. I examined her thoroughly but could find nothing. As the child was of an exceedingly nervous disposition and had dreaded the dressings to an unusual degree, I was inclined to think the night crying was due to her nervous temperament.

Two days later, she suddenly developed a temperature of 104°, with a pulse of 60; rigidity of the neck and marked Kernig. The blood count was WBC 37,000; polys., 84%. The spinal fluid was positive for streptococcus hemolyticus. Blood culture, negative. The child died in a few hours.

In this case, death occurred six weeks after the double mastoid operation and two weeks after the wounds were completely healed. The culture from the mastoid wounds showed streptococcus, short-chain; the culture from the spinal fluid, six weeks later, showed streptococcus hemolyticus. Aside from the night-crying for a short period only, the child had been apparently perfectly well for the preceding two weeks.

CASE II. (Nine weeks).

History. Mrs. E. G. B., aged 29, was admitted to Mountainside Hospital, on the 29th of April, 1921. She gave a history of a severe cold in the head which had developed two days previous to admission; severe pain in the right ear, behind the right ear and over the right eye and cheek, associated with dizziness, developed twelve hours previous to admission. The right ear broke about twelve hours before admission and had been discharging since. Her temperature was 101.2°, pulse 100, respirations 22.

Physical examination showed her to be a well-developed woman who appeared to be extremely prostrated. There was considerable tenderness over the right frontal and maxillary sinuses and the right mastoid. The right side of the nose was filled with pus and completely occluded by the swollen turbinates. There was pus in the middle meatus on the right side of the nose and the right antrum transilluminated dark. There was profuse, thick, yellow discharge in the canal of the right ear, with a fundus considerably narrowed downward. Hearing on the left side was normal, in the right ear, she could hear nothing. There was no response to douching in the right ear. A spon-

taneous nystagmus to the left was present. X-ray showed the right mastoid very cloudy throughout, with considerable breaking down of cells. The right antrum was also cloudy.

Laboratory findings: Urine, negative. Blood count-Hemoglobin 68%; RBC 3,370,000, WBC 38,000; lymph., 7%, polys., 90%, trans., 2%. Culture from the aural discharge showed streptococcus hemolyticus. Temperature just before operation went to 104°.

Operation. Operation disclosed a very large, deep mastoid, the cells of which were filled with pus and granulations. No sinus or dura was exposed. The sinus was far forward. The cells ran deeply into the petrous bone, beneath the labyrinth, and above the jugular. At the time of operation the right antrum was washed out and an ounce of thick pus obtained. Culture from the mastoid wound showed streptococcus hemolyticus. Blood culture was negative.

Clinical Course. The day following the operation, the patient's temperature dropped to 101.3° and went up to 102.4°. The vertigo was better and there was no vomiting. Next day the temperature went to 99.3°, and was normal the fourth day. She still had spontaneous nystagmus to the opposite side, slight vertigo, no vomiting. Her temperature remained around normal for the rest of her stay in the hospital (nine days). Outside of a slight unsteadiness in gait, a slight spontaneous nystagmus to the opposite side, and an occasional sharp pain in the wound, she had no unpleasant signs or symptoms and was allowed to go home. At this time she had a dead labyrinth; there was no pastpointing; the eye-grounds were negative.

Three weeks after the operation, the wound had almost completely healed. There was no spontaneous nystagmus. She complained of slight pain in the wound at times and felt slightly dizzy on occasion. The labyrinth still failed to respond to any stimulation. There was no spontaneous pastpointing.

Seven weeks after the operation the drum membrane was normal in appearance, the mastoid wound very nicely healed. She still complained of occasional pain in the wound. There was no pastpointing, no headache, no nystagmus, no ataxia, vertigo or astereognosis. The labyrinth was dead to all stimulation.

Nine weeks after the operation, she awoke one morning with severe pain in the wound which was attributed to lying on the ear. Later in the morning, she felt dizzy and vomited; this was thought to be due to an upset stomach. That afternoon she was seized with a general convulsion, at which time I was notified of her condition. She died two hours later before I reached her, respirations ceasing before the heart.

Up to the morning of the day she died, the patient had been going about, attending to her household duties. She had been at the hospital with her child three days previously, while he had his tonsils and adenoids removed. The evening before, she had been out driving and had stated that she felt unusually well.

Death occurred in about twelve hours from the onset of meningeal symptoms, nine weeks after mastoidectomy and six weeks after the wound had healed. The culture from the mastoid wound at the time of operation was streptococcus hemolyticus; culture from the spinal fluid and from the blood at the time of death was pneumococcus group III. Blood count at the time of death was WBC 32,000; polys., 96%.

CASE III. (Three months).

History. C. B., aged 19, was admitted to St. Luke's Hospital, New York City, on September 9th, 1921, with a history of discharge from the left ear for three weeks, accompanied by considerable pain in the left temporal region and the left eye.

Physical examination showed a well-built, well-nourished boy, decidedly ill in appearance. The left eye was swollen and the conjunctiva congested. He was normal in every other way with the exception of the left ear.

There was thick yellow discharge in the left auditory canal; the fundus was very narrow. There was slight mastoid tenderness. He could hear a loud whisper three inches on the left side. The right ear was normal in every way. There was a slight spontaneous nystagmus to the left. X-ray showed a very cloudy left mastoid, with some breaking down of the septa. His temperature was 102°, pulse 92, respirations 22. Blood count gave WBC 14,500; lymph., 32%; polys., 68%. Urine, negative.

Operation. Operation showed an exceedingly large cellular mastoid requiring a large posterior incision. The cells were all necrotic. There were large zygomatic cells running far forward and for about half an inch above the meatus. There was a large emissary vein over the sinus and above the antrum. No sinus or dura was exposed. Culture from the mastoid wound unfortunately was lost.

Clinical Course. In the course of the next three days the temperature gradually came down to normal. The patient, however, complained of continued pain in the left temporal region and in the left eye. This eye was still swollen and the conjunctiva remained red. He stayed in the hospital for two weeks during which time his pulse was as low as 65 on four or five occasions, but averaged about 80. The temporal pain gradually improved but was variable in intensity. The slight spontaneous nystagmus to the left was very variable. The eye-grounds and pastpointing tests were negative. There was no rigidity. He complained of considerable tenderness of the scalp over the entire left side of the head. There was considerable discharge from the left meatus but the mastoid wound was clean and there was no swelling.

At the end of two weeks, he had no nystagmus; no evidence of cranial nerve involvement; no aphasia; the eye-grounds remained negative. The conjunctiva of the left eye continued red. The left side of the head was still tender. He still had pain in the left temporal region but at night only. He was, however, able to sleep all night without medication. He was allowed to leave the hospital.

During the following eleven weeks the wound healed up with the exception of a small fistula an eighth of an inch in diameter, which at times was clean, at others contained a drop of pus. Discharge from the ear persisted, coming from a tit-like protrusion in the upper part of the drum membrane. During this time he would be free from headache for a period of ten days and then might have a severe headache lasting a few hours. Nystagmus to the left was present on and off. He had no cranial nerve weakness, no ataxia, pastpointing or vertigo. His temperature remained normal, his pulse about 70. The eye-grounds were negative. The hearing on the operated side was five-twentieths. He returned to college, took up his work there and appeared perfectly normal, excepting for an occasional temporal headache and the fact

that his wound would not heal up and that the discharge from the meatus persisted.

Fifteen weeks post-operation, he suddenly began having a very severe temporal headache and vomited several times. There was no nystagmus, no ataxia, no cranial nerve weakness.

Secondary Operation. I persuaded the parents to let me explore the mastoid region and examine the dura in the middle fossa. This I did, finding nothing. I removed all the bone over the middle fossa for an area of one and a half by one and a quarter inches. The dura looked slightly grayish and lustreless but was under no tension and no fistula could be found. I thought possibly there might have been a collection of serum under the dural plate and hoped that this might relieve the headache.

Clinical Course. During the following four days, the patient felt better, was up and about. He still had a slight headache at times but said he felt much better than before operation. The eye-grounds were negative and there was no nystagmus. Spinal puncture was not attempted because the parents objected to anything being done that was not absolutely necessary. On the evening of the fourth day after the operation, he discharged his night nurse and spent the evening playing cards. He had been up and about the hospital and was planning on going to a hotel the next day.

About two o'clock that night he became delirious and died fourteen hours later of a meningitis due to a pneumococcus group III. Death occurred three months after the mastoidectomy. Blood count just before death was WBC 20,000; polys., 90%. Blood culture was negative. Spinal fluid showed pneumococcus group III.

Remarks.

Case I showed a short-chain streptococcus in both mastoid wounds; six weeks later,, three weeks after the wounds were healed, streptococcus hemolyticus was found in the spinal fluid, with a negative blood culture.

Case II showed streptococcus hemolyticus in the mastoid wound; nine weeks later, six weeks after the wound had healed, there was a pneumococcus group III in spinal fluid and blood.

Case III, culture from the mastoid wound was lost; fifteen weeks later the spinal fluid showed pneumococcus group III.

Two of these cases died of pneumococcus group III meningitis; one of streptococcus hemolyticus. One of the pneumococcus meningitis cases had pneumococcus group III in the blood; she also had a suppurative labyrinthitis. The other meningitis case had a negative blood. Two of them developed fulminating meningitis weeks after the mastoid operation. Two of them weeks after the wounds were healed.

As is well known the meninges, the middle ear and the mastoid are all involved occasionally, at the same time through the blood. It

is claimed by some that all pneumococcus meningitis cases are infections through the blood and that the mastoid wound has nothing to do with it. In these cases the only meningitis which may have come through the blood was the first case, and that was a streptococcus hemolyticus infection. The other two meningitis cases were due to the pneumococcus group III. One of these had a suppurative labyrinthitis, the other had a discharging wound. Cases with histories very similar to these cases have been reported where at autopsy a pachymeningitis was found in the region of the mastoid. These cases all had a pneumococcus group III infection. They all developed their meningitis symptoms weeks after the wounds were healed. The last two cases of this paper undoubtedly belong to this group and not to blood infection. Whether the meningitis is due to blood infection or to mastoiditis is a very important question to decide as operative procedure is useless in blood infection and may be important in wound infection.

The fact that two different organisms were found in the mastoid wounds and spinal fluid is hard to explain whether blood infection or not.

Any mastoid case who complains of pain in the operated side, no matter how trifling and transient should be watched with great care and regarded with grave suspicion, for many weeks after operation, especially if the infecting organism is the pneumococcus group III.

Pneumococcus group III in an aural discharge calls for an earlier operation than the other infections.

Rupture of Carotid Artery: Result of Chronic Mastoiditis.

History P. B., aged 38, an Italian laborer, was admitted to Bellevue Hospital, New York City, on November 20th, 1918, complaining of severe pain in the right ear. The history, obtained from the patient's sister, was that he had had discharge from both ears for many years. Eight years previously he had a "boil" back of the ear. (This "boil" was in all probability, a cortical performance of the mastoid). At the same time he was said to have had typhoid fever and pneumonia.

Physical examination showed a poorly nourished Italian who appeared acutely ill. Outside of the ear condition he was normal, physically. The temperature was 101°, pulse 88, respirations 24. He seemed unusually stupid and dull; had periods of restlessness and at times would have to be roused. The right ear showed a profuse discharge; the drum was reddened and thickened, with a perforation anteriorly; there was some sagging of the posterior-superior wall. He could hear nothing and caloric was negative.

Laboratory findings: Urine, negative. Blood count, WBC 13,300; polys., 81%, lymph., 3%, trans., 5%, large monos., 5%, eos., 2%, myel., 4%. X-ray

showed the left mastoid cloudy, with a partial obliteration of the septa. The right mastoid was markedly cloudy with sclerosis of the mastod cells. Previous to operation his temperature ranged from 98° to 103°.

Operation. A right, radical, mastoidectomy was performed. A large cholesteatomatous cavity was found extending from the dural plate, anteriorly, to and including the middle ear; posteriorly, it extended to the sinus plate. A large abscess was also found in the tip of the mastoid. Only a small portion of the posterior canal wall remained and this fell away in one large sequestram exposing the facial nerve which was held in a mass of necrotic bone. This bone was carefully removed, allowing the nerve to be free in the cavity. The dura was exposed above the antrum. No evidence of any of the semicircular canals was found. The middle ear was filled with granulations. A blunt probe was inserted downward in the middle ear in order to ascertain how much overhang. The probe met no resistance for a distance of half an inch and pus seemed to be welling up under pressure. This being a very dangerous locality, I decided to drain this area and later attempt to clean it out. The wound was therefore packed wide open.

Subsequent Course. About four hours after the operation, there was an unusual amount of oozing through the mastoid dressing and the patient expectorated some blood. The dressing was changed, no bleeding points found and the wound packing was reinforced. The following day the patient again bled from mouth and nose and the mastoid wound was repacked. Twenty-four hours later, two days after operation, the house-surgeon was again called because of bleeding from nose and mouth. This time the flow was more profuse. The house-surgeon told the man to rinse his mouth with some ice-water. On his so doing, there was a tremendous flood of bright red blood from mouth and nose. In forty seconds the man was dead. Immediately thereafter, the house-surgeon explored the nasopharynx with his finger. In the neighborhood of the Eustachian tube, the tissues were all necrotic, so that his finger easily entered them, meeting no resistence.

This, undoubtedly was a case of necrosis of the carotid artery. Should I ever again encounter so much destruction in the floor of the middle ear, I would think seriously of tying the carotid artery immediately. It would have been impossible to have interfered any less or to have been any more careful in my manipulations in the middle ear.

DISCUSSION.

Dr. Edward B. Dench, New York City, said that he had listened with a great deal of interest. In regard to the procedure mentioned by Dr. Bowers; he spoke of interfering with the sinus before the jugular. The speaker thought it better to ligate the jugular first and avoid possible infection of the blood stream. He used to do the other way first but had had a change of heart since.

In reference to the facial paralysis occurring on the opposite side of the lesion,—the explanation was probably toxic. It would be interesting to know whether there was pain on the opposite side; that would make the toxic explanation more easy. The cases at St Luke's had no nystagmus. Dr. Bell had cases with nystagmus, they looked like neuritis of the geniculate ganglion. Some cases are no doubt interference with the optic nerve from increased intracranial pressure. With lumbar puncture the cases clear up more rapidly.

Cases two and three, had streptococcus capsulatus as the cause of infection. Dr. Dench recalled two or three of these cases, apparently cured,

and then later developing sudden meningitis followed by fatal termination. The middle cranial fossa was explored. It was negative. In one case recalled, the man had acute middle ear infection 6 weeks, with extensive destruction of the mastoid process. The sinus had been completely destroyed. Dr. Dench incised the internal jugular. There was no hemorrhage from the bulb. It came from the other end. The man appeared to be doing well, but later the house surgeon called up to report he had passed into coma. He died within 24 hours. Such cases are discouraging. One can say that group III cases are not safe under a year post-operative. One has to watch these cases like malignant disease, and can only conjecture how they occur. Possibly the organisms may become isolated by pial adhesions in the cerebrospinal system, then suddenly become liberated and the fulminating meningitis is the result. Where mastoid operation has been done, certain organisms are completely walled off. This is what Eagleton calls protective meningitis. When there is a break, the germs are thrown into the circulation, and death results. It is a hopeless situation. Dr. Dench said he was glad to hear Dr. Bowers speak of the extensive destruction in the tympanic cavity. Dr. Bowers had anticipated him. He thought that putting a ligature around the internal carotid would be excellent surgery.

Dr. Wendell C. Phillips, New York City, said that he would emphasize the importance of continuous headache in mastoid operation or operation for acute otitis media. He had seen many patients give this sign in serious brain lesions ending in fatal meningitis. The only symptom was persistent unilateral headache. He recalled a chronic suppurative case two years ago with very large cholesteatoma. The speaker said he made wide exposure of the dura. The man complained of headache following operation. Every conceivable examination was made, and it was supposed that it was an encapsulated brain abscess. The man went home. Two months later the doctor was suddenly called up to see the man who had gone into coma, and in 24 hours had died of meningitis. Headache would seem to be the only symptom until the cases are far advanced.

Dr. L. W. Dean, Iowa City, said that a week ago a child had entered his service with neglected acute left mastoid. Blood culture was negative; temperature not over 100°. The child was not very sick, was walking about and showed a paralysis of the 6th and 7th of the opposite side. The X-ray was negative; the neurological report was paralysis of the 6th and 7th due to ear condition on the opposite side. The school teacher said the side of the child's face had bothered him and that turning of the eye occurred at the same time. Unfortunately no Wassermann report of the blood and spinal fluid had been obtained to date. Unless the case proves to be syphilis plus toxemia, it will be very difficult to understand, as operation revealed necrosis of the mastoid without any sinus involvement.

Dr. Perry Goldsmith, Toronto, Canada said that a German prisoner, with a bullet wound behind the ear, had become unconscious, delirious, with slow pulse and vomiting. There were all the symptoms of brain abscess. The bullet did not break down the mastoid but passed through the ear and produced fracture of the petrous portion of the temporal bone with mastoiditis.

Dr. J. F. Fairbairn, Buffalo, N. Y. said that autopsy report on these cases would reveal far more than was known at present. He had one case of a man with persistent headache, after operation on a large pneumatic mastoid. Operation was in January. He went home in March. The spinal fluid had shown pneumococci. When the wound was open and draining the headache disappeared. When the wound closed headache persisted. There was necrosis of the tip of the temporal bone. Probably the lesion extended upon the anterior surface of the temporal bone, toward the apex.

Dr. J. W. Murphy, Cincinnati, O. said that he had autopsied nine cases. In the pneumococcus III cases, there was low grade virulence, because of the capsule. There were four cases in the ward three weeks with normal blood count, then they became unconscious, and there was high leucocytosis. At autopsy the fluid was thick with gelatinous exudate. About 12 percent. of the cases seen are from type III pneumococcus. The germs are hard to identify on culture. In regard to the cases where streptococcus is said to be found in the mastoid and pneumococcus in the blood stream, the speaker said that is due to the bacteriological reactions, which are misleading, as it is very hard to differentiate the two types culturally.

Dr. Wesley C. Bowers, (closing) said that he believed in tying the jugular, even before blocking off the sinus. He did not know why he had reversed procedures in the two cases cited. Continued headache is a very important point. He was suspicious of the patient because of continuous pain in the temporal region and swollen left eye. He was certain there was abscess in the dura, but on opening the dura found nothing. Autopsy could not be obtained. The Wassermann was negative for blood and spinal fluid. There are cells running down into the petrous bone which may play a part in the extension of the petrous abscess into the dura. This would be a good explanation of the process.

A CONTRIBUTION TO THE STUDY OF BRAIN ABSCESS.*

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In reviewing the subject of brain abscess one is struck by the scarcity of symptoms observed. Most of the case histories I have been able to study are somewhat meager, and in many cases one cannot assume that the lack of specific mention of certain symptoms means that they were absent. They were probably overlooked. Yet, even in the cases where positive and negative findings are definitely stated there is usually relatively little upon which to base a definite diagnosis. Still, there is generally some indication present if all sources of information are explored. The clinical picture of brain abscess presents a wide variety of symptoms and signs, but this picture is a composite one, and one can never expect to find more than a few symptoms in any one case. Symptoms of increased intracranial pressure are often fairly definite. These, however, may be due to extradural abscess, brain abscess, tumour, or meningitis. The more acute the condition the more violent are the symptoms. Thus, in tumor, which generally takes months to grow, although the mass may be large, signs of increased pressure may not be very pronounced. In the case of abscess they are generally more marked, and still more so in the case of meningitis, which may develop in a few days.

In this paper I propose to give an analysis of the notes of sixty-seven cases of otogenic brain abscess obtained from the literature.

Frequency of Occurrence.

From figures obtained from the Edinburgh Royal Infirmary Reports the frequency of intracranial complications is 2.19% of all cases of middle ear suppuration. Brain abscess occurs in 1.38% of all cases of middle ear suppuration, or in 4 per 1,000. Intracranial complications occur more than twice as often in chronic middle ear suppuration as in acute. Mygind out of 207 cases of intracranial complications found meningitis in 68%, sinus phlebitis in 50%, brain abscess in 20%, and subdural abscess in 9%.

*Candidate's Thesis recommended for publication by the Council.

Tracks of Infection from Middle Ear.

In the case of temporosphenoidal abscess probably by far the greater number are caused by direct spread of infection upwards through the tegmen tympani. In most of the recorded cases which were available for study, the tegmen was found to be eroded and the dura covered with granulations. In such cases a localized patch of meningitis is set up, the pia arachnoid becomes adherent to the dura and the process spreads into the brain itself, forming a subdural abscess. Vessels on the surface of the brain become thrombosed and cause septic softening in the white matter. In other cases however, the tegmen is not eroded and when the dura is exposed it appears healthy except for increase of tension. The brain is not by any means always adherent to the membranes when there is an abscess underneath. The surface frequently looks quite normal. Here it is more than likely that the abscess originates in a small septic embolus. That part of the temporosphenoidal lobe which lies over the tegmen represents one of the boundary zones in the cerebral circulation, where the regions supplied by the middle and inferior cerebral arteries meet. The vessels here are hence all of small calibre and easily permeable.

Another factor in the etiology of brain abscess is the possibility of infection traveling along thrombosed veins or venous sinuses. Some of the blood from the middle ear drains through the subarcuate vessels into the superior petrosal sinus. Numerous veins from the temporosphenoidal lobe and also from the front part of the cerebellum also enter the superior petrosal sinus. Other vessels from both parts of the brain enter the lateral sinus just about the genu. A case has been described by Fraser (1) in which infection passing along the subarcuate vessels was demonstrated microscopically. It is quite probable that a small thrombosis may be formed in one of the cerebral or cerebellar veins and the infection in this way reaches the brain. In this connection it is interesting to note that in 43 temporosphenoidal abscess cases sinus thrombosis was present in only four, and perisinus abscess in only one. In the 24 cerebellar cases on the other hand thrombosis was associated in 12 instances, while in three others there was a perisinus abscess without thrombosis. This rather points to the probability of venous embolism as an etiological factor in the formation of

abscess of the cerebellum, though not necessarily of the cerebrum.

Frequency of Complications.

According to Milligan (2) the infection reaches the cerebellum in acute cases through extradural abscess or sinus thrombosis. In chronic cases the abscess is generally secondary to labyrinthitis.

In this series in roughly half of the temporosphenoidal cases the abscess occurred without other complications, such as extradural abscess, sinus thrombosis, or meningitis. In cerebellar abscess on the contrary only 3 out of 17 were found without any other complications.

Injury as a Causal Factor.

Five of the cases under review had received some injury to the head sufficient to cause loss of consciousness. This appeared to play some part in the formation of the abscess, as the symptoms dated from the injury in most of them.

In some cases the mastoid operation appeared to act as the exciting cause in the formation of the abscess. In several other cases, however, in which symptoms were present leading the surgeon to suspect brain abscess the mastoid operation was followed by complete disappearance of the symptoms for some weeks. When they finally reappeared they were accompanied by signs of meningitis or rapidly spreading encephalitis.

Cause of Death.

In a study of 25 fatal cases of brain abscess it was seen that diffuse purulent meningitis was the cause of death in 12. In two of these the meningitis had been set up by leakage from the abscess into the subarachnoid space. Five cases died from pressure symptoms, in three of which death was due to acute encephalitis or edema. In three cases the patients died with pressure symptoms due to a second undiscovered abscess. In three death was due to rupture of the abscess into one of the ventricles. In my own experience this latter complication is generally fatal within a few hours, but in one of Fraser's cases death did not occur till 17 days after rupture.

General Symptoms of Brain Abscess.

Brain abscess is generally described as showing three clinical phases, (1) an initial, (2) a middle, and (3) a terminal stage. The first stage is very seldom seen in hospitals. It corresponds with the formation of an area of red softening and is sometimes ushered in by a chill, headache, and vomiting. The chill may be merely a feeling of cold with cutis anserina, or may be an actual rigor. The temperature may be slightly raised. In the middle stage the signs of increased pressure predominate. The senses are blunted, the cerebration is slow and the mind is dull.

Headache is severe but does not cause outcry. The pulse and respiration rates are slow and the temperature is normal or subnormal. The terminal stage is short and is characterized by coma, incontinence, and increasing respiratory failure. Hyperpyrexia and frequent pulse may immediately precede death.

Encephalitis, abscess, and meningitis give a series of symptoms which are common to all but vary in degree. The symptoms are in the main due to the accompanying increased intracranial pressure. These are headache, vomiting, mental dullness, drowsiness, optic neuritis, slow pulse, and often subnormal temperature. MacEwen described chills as one of the early symptoms of brain abscess, but in many of his cases there was sinus thrombosis or perisinus abscess as well which may have been the cause of the chills. In this series out of 67 cases in all, rigors occurred in 15, and of these 15 nine were cases with sinus thrombosis. From this it would appear that rigors or chills sometimes occur with simple brain abscess, but that in most cases where such a symptom is noted it is due to an accompanying sinus thrombosis.

Headache is one of the most constant symptoms and was present in 50 of the 67 cases, while in some of the remaining cases its presence or absence could not be ascertained as the patient was already comatose. Where details were given the headache had generally commenced on the affected side and was extremely acute, often enough to prevent sleep. Later it was generally frontal or occipital or both.

Mental disturbance manifested in various ways has been described by different observers as slowness of cerebration, obnubilation or clouding of the intellect, apparent stupidity, absent-mindedness, lack of attention, psychic depression, etc. In several instances the mental condition was practically the only symptom

of the abscess. Adson (3) notes symptoms of mental disturbance in 15 of his 26 cases. This clouding of the intellect, inability for sustained attention, or for any exertion are due partly to the increased intracranial pressure and partly to the amount of toxin in the cerebrospinal fluid.

Drowsiness is not found in the earlier stages as a rule, but only in the second and final stages. As the intracranial pressure increases so does somnolence till it merges into coma. In the second stage the patient generally is half asleep but he can be roused and will answer questions, but immediately drops off to sleep again.

Vomiting. In the cases under review it was noted as a symptom thirty times. However, it was doubtless present in many more of the cases, as in those which were carefully detailed it was always mentioned. The characteristic of vomiting due to brain abscess is that it bears very little relation to the taking of food, and is usually unaccompanied by nausea. Projectile vomiting, so-called, is not very common. It was noted as such in only one case. The vomiting in the initial stages of brain abscess and sinus thrombosis and meningitis frequently accompanies a rigor. Moving the patient in bed during an examination very often brings it about.

Nausea, which is so marked a feature of labyrinthitis, is generally absent in cerebral abscess, though sometimes present in cerebellar abscess.

Obstinate constipation is a frequent symptom when the intracranial pressure becomes great. The tongue is also often very thickly furred.

Optic Neuritis. Adson found choked discs in 9 out of his 26 cases. Logan Turner (4) in 14 cases of intracranial complications of otitis media found optic neuritis in none of them, though in some there was blurring of the edges of the discs and engorgement of the vessels. The condition of the eye grounds was noted in 34 cases in this series and in 26 of them there was some change, and the condition was generally more marked on the affected side. Optic neuritis is not as a rule seen in cases where the increase of intracranial pressure had lasted less than several weeks. It appears earlier and is more severe in posterior fossa infections than in those of the middle and anterior fossae. Blindness is said to be much commoner as a result of cerebellar than of temporosphenoidal abscess. According to

MacEwen the optic neuritis is rarely followed by atrophy and the sight generally returns when the brain condition is relieved. To summarize, the absence of optic neuritis does not rule out the diagnosis of brain abscess, but its presence is an important confirmatory sign.

Pupils. In the early irritative stages the pupils are contracted and active. When pressure symptoms begin to predominate they tend to dilate and are sluggish in their reaction to light. The state of the pupils was noted in 21 cases. In five of these both pupils were dilated and fixed, and in all of them other pressure symptoms were very marked. In six they were equal and small, and in all of these except one pressure signs were not severe. In ten instances the pupil on the affected side was dilated and in most of them it was fixed. MacEwen states that when the abscess is in the frontal or temporosphenoidal lobe the pupils may be either small or large, but especially sluggish. Sometimes the only difference is that the pupil on the affected side is more sluggish.

Pulse. In the initial stage of brain abscess the pulse may be frequent, but in the stage when cases are usually seen the pulse rate is generally slow. A pulse rate of 50 to 70 is common, and in this series there were four cases with rates between 40 and 49. This tendency to remain subnormal is generally very little affected by accompanying complications or intercurrent disease. When the abscess ruptures into the ventricles or on to the surface the pulse rate and temperature rise suddenly and continue elevated till death.

Temperature. It is more difficult to generalize on the question of temperature as there is more variation than with the pulse. When other pressure signs are present a reading of 96-97 F. is common. However, when meningitis exists as well, the temperature may remain above normal and the signs of abscess be completely masked.

Respiration. The respiration rate is generally about normal with a tendency to slowing. When the pressure is great and the patient is becoming comatose the breathing tends to become intermittent and often of the Cheyne-Stokes type. When death from increased pressure is approaching the respiratory function is usually the first to go.

Leucocyte Count. Dauling (5) studied the cytology of the blood in a large series of ear cases. His results are briefly as follows:

in acute middle ear suppuration with mastoiditis the leucocytes averaged 12,000, chronic middle ear suppuration with acute mastoiditis 17,600, extradural abscess 18,400, brain abscess 12,000, meningitis 25,500, and sinus thrombosis 17,000. The administraton of an anesthetic sends the leucocyte count up several thousands for a few days.

From the foregoing it is evident that the leucocyte count does not help materially in the diagnosis of brain abscess. In obscure cases in which one is uncertain as to the presence or absence of a latent abscess, the mastoid condition alone would give a blood picture very similar to that of brain abscess. However, as one can exclude extradural abscess and meningitis by other means, and if the uncertainty occurs more than five days after operation or in the absence of acute mastoiditis, a leucocyte count of over 12,000 would suggest the presence of abscess.

Cerebrospinal Fluid. Adson states that when there is no diffuse encephalitis or meningitis the cell count in the cerebrospinal fluid is normal in brain abscess. Fleischmann (6) in an extensive series of examinations of the cerebrospinal fluid in ear cases, of which 12 were brain abscess, found the pressure increased in most of the cases though not in all. In most of them there was a marked increase of lymphocytes up to between 30 and 300. The total albumin and globulin were increased, though not constantly nor to a marked degree, the normal albumin content being about 0.2 to 0.5%. Sugar is present in brain abscess cases, but is generally absent in meningitis. The change in the number of cells is the most constant sign.

Lumbar puncture had been performed in 24 of the cases in this series one or more times. The results were so variable and the points noted and omitted so different that one cannot draw any very definite conclusions, except that the pressure was in most cases raised. In those cases where the cell count was mentioned it was increased, and in quite a number the increase was enough to cause turbidity. In other cases the fluid was clear but contained cocci of various sorts. While the presence of germs in the cerebrospinal fluid must always be regarded as an ominous sign, these cases are by no means always fatal, as there were four in this series alone which recovered.

Let us now consider the symptoms and signs which help us to determine the site of the abscess. When the dura is exposed by disease one naturally expects to find the abscess in the ad-

joining part of the brain, but when there is no guide of this sort one has to rely entirely upon the results of neurological and psychological examination.

Localized Pain. MacEwen draws attention to the value of percussion of the head in locating the site of the abscess. Patients with brain abscess generally resent tapping over the region of the abscess, while they do not mind it over the rest of the skull. Adson found local tenderness in 12 of his 26 cases, and in 8 of them it was over the position of the abscess. In several of the other cases reviewed here tenderness to percussion was noted.

Temporosphenoidal Abscess.

In addition to the above general symptoms and signs temporosphenoidal abscess often shows some localizing signs, such as paralysis on the opposite side, convulsions, aphasia, etc. In 43 cases of temporosphenoidal abscess hemiplegia or paresis on the opposite side was noted in seven instances. Paralyses are less common in cerebellar abscess, but when present they occur on the same side as the abscess, in contra-distinction to temporosphenoidal abscess. Weakness of the arm of the same side was noted in three cases only. When paralysis of the opposite side is present in suspected temporosphenoidal abscess it is important to note the order of involvement of the limbs. If face, arm, leg is the order of involvement the chances are that we are dealing with a subdural abscess or meningitis. If in the opposite order it is more likely to be due to pressure on the internal capsule. If the infective process extends as far as the basal ganglia hemiataxia, marked disturbances of deep sensation, and slight hemiparesis, and possibly choreic movements may be found.

Paralysis of the cranial nerves is more commonly due to meningitis than to brain abscess, but paralysis of the third, fourth, or sixth nerves of the same side occurs from pressure of a swollen temporosphenoidal or cerebellar lobe.

Reflexes. The reflexes are usually very little altered unless there is already paralysis.

Convulsions. Adson reports Jacksonian fits in 12 and grand mal epilepsy in 4 of his 26 cases. In this series convulsive movements of the limbs were noted in eight of the 67 cases, and of these 7 were from temporosphenoidal abscess. Convulsions or jerkings are therefore not very common in uncomplicated brain abscess, but when present are of considerable value in localizing the condition. Twitchings are rare in cerebellar abscess.

Disturbance of Speech. Anarthria or motor aphasia is associated with the lenticular zone of Pierre Marie, which he defines as including the central grey nuclei, the internal and external capsules, the insula, the motor cortex, and some of the important underlying white fibres. The third frontal convolution is not included. Anarthria is not confined entirely to the left side of the brain, but is also found in corresponding lesions of the right side.

True aphasia is associated exclusively with the left side of the brain in right-handed individuals. It is located in the area of Wernicke, which includes the gyrus supramarginalis, gyrus angularis, the posterior end of the two first temporal convolutions and the underlying white matter as far as the wall of the descending horn of the lateral ventricle, which immediately adjoins the already mentioned lenticular zone. Lesions of the left temporo-parietal region in the area of the supramarginal gyrus produce aphasia with some anarthria. Lesions in the middle and posterior parts of the temporal convolutions produce a difficulty in naming objects. The intellectual faculties are very much affected, especially that of calculation. A lesion in the left posterior temporal and angular convolutions produces alexia predominantly, frequently associated with hemianopsia if the lesion is deep. Dysarthria or difficulty of speech may also be caused by cerebellar or bulbar lesions.

In 31 temporosphenoidal abscesses there were 15 on the right side and 13 on the left side, while in 3 the side was not stated. Aphasia was noted in 7 of the 13.

Cerebellar Abscess.

Cerebellar abscess is much richer in diagnostic signs than cerebral abscess. Among the symptoms and signs are nystagmus, vertigo, troubles of balancing and coordination, including falling, pointing error, ataxia, loss of muscular tone on the affected side, intention tremor, etc. Repeated yawning and rigidity of the masseters were frequently observed by MacEwen.

Nystagmus, apart from that of ocular origin, which is easily distinguished, is almost invariably due to a lesion of an irritative or destructive character either in the labyrinth or in the posterior cranial fossa. As a rule there is no great difficulty in differentiating between a labyrinthine and a cerebellar lesion, but when latent labyrinthitis and cerebellar abscess occur concomitantly the actual state of affairs can only be diagnosed by a very detailed examination and careful analysis of the signs. A

detailed discussion of these would require too much time and space to be entered into here, and I am afraid I have already exceeded my time allowance.

In conclusion I should like to state my conviction that cases of brain abscess, though difficult to detect on account of the want of obvious symptoms may be revealed in almost all instances by thorough and repeated neurological examinations. Taking the obscurity of the condition into consideration we cannot afford to dispense with any of the present diagnostic methods, but in every suspected case should subject the patient to an absolutely complete examination, including psychological, neurological, blood and cerebrospinal fluid examination, etc.

I would go further and state that every patient before undergoing a radical mastoid operation should have a moderately complete neurological examination.

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DISCUSSION.

Dr. I. Friesner, New York City, said that he had only seen one brain tumor in which the symptoms appeared suddenly. Dr. Dickie mentioned sinus thrombosis was associated with 12 out of 24 of brain abscesses. That is a high proportion, usually 25 per cent. was noted. Drowsiness is interesting. He had not seen many cases in which there was increased tension with disturbance of the sensorium. Toxicity has probably more influence than pressure. The presence of cocci in the fluid should be regarded with suspicion unless there is cultural growth. Often artefacts from straining reactions have the appearance of cocci. In brain abscess there were often 100 per cent. lymphocytes and many cells of the fluid.

Dr. Lewis Fisher, Philadelphia, Pa., said that Dr. Dickie placed more reliance on the neurological findings in brain abscess than he had been able to do. He had rarely found definite neurological signs. In one case in which abscess was suspected for weeks, there were no signs till two days before death. Unless the abscess happens to be located in a definitely known brain area the neurological examination is apt to be negative. Comparison of early and late findings will help in differentiation. Absence of response, from the verticle canals suggests that pressure is interfering with response, and intracranial complications can be looked for. When the horizontal canals are functioning there is probably a pathological reason for the difference in function of the two sets of canals. In cerebral abscess the patient will not as a rule tolerate touching the spot over the abscess. In cerebellar abscess rotation tests show reduced duration of the vertigo.

Dr. George MacKenzie, Philadelphia, Pa., said that symptomatology of brain abscess is so rich that it hinders differentiation. Usually there are contracted pupils in brain abscess, due to spasmodic myosis. Every case of mastoid trouble should have an exhaustive examination at the hands of the otologist. When one makes a diagnosis of middle ear disease, one has done no more than to confirm what the patient says. Labyrinthine conditions must be diagnosed, and when brain abscess is noted, the tract of infection, whether through the sinus, or posteriorly must be stated. In brain abscess the condition of the meninges and the internal ear must be known. In cerebral abscess there is slow pulse, low temperature and slow (sometimes suspended) respiration.

Dr. Isaac Jones, Los Angeles, Cal., recalled a case of a boy 12 years old with a running ear, in whom everything was negative, except that at autopsy a pin point lesion found below Shrapnel's membrane. There was no discharge, no pain, no tenderness. He had marked disturbance of the vestibular response, spontaneous vertical nystagmus, and also pressure nystagmus. Dr. Ingham made a diagnosis of the right cerebral hemisphere. The abscess was found there. The boy did well for a time autopsy revealing that abscess was limited and that death occurred by improper walling off round the drainage tube, from which there was spread of infection to the meninges. There was a mastoiditis which had never given any symptoms. In regard to nystagmus,—cerebellar lesions produce nystagmus because of involvement of the brain stem. Dr. Eagleton mentioned variability of the signs. In this case the boy had a large vertical nystagmus looking upward, but always present. Operation with relief of the abscess caused disappearance of the nystagmus.

Dr. S. D. Ingham, Los Angeles, Cal., said he was surprised to see what good neurologists the otologists were. Their work was closely related, but the questions were approached from a different angle. The paper was a most complete presentation of the subject. Abscess of the brain could be put in the same class as tumors. Dr. Jones mentioned one case diagnosed tumor where abscess was found at operation. The boy was a Mexican, and there was no history. He had symptoms of cerebellar disturbance. Diagnosis of tumor is a difficult matter, those of large size can exist with few symptoms. Even gliomata which involve the well known areas can exist without causing symptoms. One case recalled had very slight symptoms of one side of the face, but severe headache and choked disc. There was a large tumor of the frontal area. In regard to Dr. Fisher's view point as a neurological findings, that is true, but the neurological findings are all one has to go by. Examination of the 8th nerve is practically a neurological examination.

Dr. Dickie (closing) said that Gordon Holmes had pointed out during the war that in localized lesions of the cerebellum the nystagmus had a tendency to come to a rest point a little to the opposite side of the middle line. He had seen one case since, and this sign was present. Labyrinthine nystagmus cannot be controlled by fixation. The movements are found to be due to a slowness in starting and stopping, such as is present in testing for adiadochocenesia. There is asynergia and the lack of harmony of the muscles causes extravagant movements.

It was a great pity McEwen's valuable work was out of print, and so hard to obtain. His findings made in 1883 had not been improved upon since.

In regard to mastoid operation setting up brain abscess,—the slight shock and traumatism, due to cutting and chiselling might dislodge infected particles and send them the wrong way. Curious complications happen sometime afterwards. In regard to cloudy fluid, there may be a little meningitis in some of the cases.

THE HISTOPATHOLOGY AND HISTOGENESIS OF BENIGN GROWTHS OF THE NOSE AND ACCESSORY CAVITIES.

By HARRY L. BAUM, M. D., Denver, Colo.*

It has been the custom of the author of this paper to give more than average attention to the science of pathology in its bearing on otolaryngology and he believes that this study of a branch of medicine somewhat removed from the usual routine of special work has been the source of much valuable information relative to the specialty and of a better understanding of many cases. As a knowledge of anatomy is essential to proper surgical skill and technic; so, in a no less definite way, is a knowledge of pathology essential to a proper understanding of diagnosis and treatment. We practice a somewhat limited specialty as otolarynologists and it is not too much to ask that we have a working knowledge of the fundamental sciences as applied to that specialty. We may not always be capable of making a histological diagnosis with the accuracy expected of one making pathology his specialty and it is not with such an expectation that attention is directed to the science, but in the hope that we may gain a clearer understanding of disease processes and of the changes taking place in the tissues with which we work.

The Edematoses. These, by far the most common pathological conditions met with in the nose and accessory cavities, have been variously called polypi, edematous polypi, myxomata, fibro-myxomata, myxo-fibromata, soft fibromata and edematous fibromata. Many writers have erroneously adopted terms descriptive of them which have not only resulted in a confusion of terminology but of understanding as to the actual histological structure of the tissues under consideration. There is nothing in the structure of a nasal polyp which justifies the use of the term myxoma as applied to it, for it contains no myxomatous tissue in its substance. Wright (49), in 1900, emphasized this point very strongly and it is also brought out in Delafield and

*Candidate's Thesis recommended for publication by the Council. Illustrations omitted for lack of space.

Prudden (16) and by Ewing in his "Neoplastic Diseases" (18), although many of the older pathologists fail to recognize this differentiation. Expressions such as "fibro-myxoma" and "myxomatous" have doubtless been used by many writers with a full knowledge of the laxity of the terms but they have failed to transmit that knowledge to their readers, with the result that there still exists a deplorable lack of uniformity of understanding and of terminology among rhinologists. The writer pleads guilty to this fault in his own case in the past and feels that a more accurate terminology should be adopted, if not histologically descriptive at least not actually misleading. To this end the general term "Edematoses" will be used in this connection for the reason that a study of the histology of these growths will reveal that the fundamental underlying change in the tissues is a serous effusion, an edema.

Simple Edematosis. To that condition in which there is simply an edema, or serous infiltration, among the fibres of the loose areolar tissue constituting the sub-epithelial layer of the mucosa and unaccompanied by demonstrable fibrous hyperplasia or marked pathological change in the tissues themselves the term "Simple Edematosis" will be applied. This classification should include such conditions as the apparent hypertrophies due to hay fever and so-called vasomotor rhinitis, many of the more lasting polypoid hypertrophies of the turbinates resulting, usually, from chronic conditions in the sinuses, and some of the earlier types of pedunculated polypi before they have undergone marked fibrous change.

Edematous Fibroma. On the other hand it is true that many of the edematoses will reveal a sufficient amount of fibrous hyperplasia to justify the name of fibroma and to these will be applied the term "Edematous Fibroma" as most descriptive. By this is meant a tumor consisting of loosely packed fibrous tissue, the site of serous effusion, containing fibrin threads, leucocytes in the form of the familiar round cell infiltration, blood vessels and at times glandular structure. In short it may contain any of the constituents of the mucosa from which it springs, to which may be added the signs of sub-acute or chronic inflammation evidenced by increase in round cell infiltration, increased fibrosis around vessels, dilatation of gland acini and in places marked fibrous tissue hyperplasia. The epithelial changes manifest themselves both as a hyperplasia of the normal

columnar and in the form of metaplastic change to cuboidal or squamous, the latter usually stratafied and frequently hyperplastic.

This classification includes the older forms of turbinate and septal edematoses and the majority of those having their origin in the accessory sinuses. At this point it is only fair to remark that the gradation from the purely edematous conditions to those justifying the use of the term edematous fibroma must be very gradual and that there must of necessity be a certain elasticity in the application of these terms. It is true, however, that at one extreme there exists the typical simple edematosis and at the other the condition of undoubted fibrous hyperplasia with edema and although histogenetically they are apparently one and the same process their evolution from one form into the other justifies the use of slightly different terminology to signify that change.

The histogenesis of this type of nasal neoplasm has been deeply studied by numerous authors, among them Uffenorde and Hajek, whose views are quoted by Marquis (33); Skillern (47), and others and it is agreed that their formation is undoubtedly dependent upon some form of irritation or inflammation in the tissues from which they spring. This results primarily in transudation of serum and later in hyperplasia of connective tissue with definite tumor formation, necessitating an accompanying hyperplasia of the epithelial covering to keep pace with the increase in substance of its underlying structures.

Pseudo Cysts. A condition not yet mentioned but frequently occuring in the development of edematous fibromata is that of so-called cyst formation. Although true cyst formation does occasionally occur in the mucous membranes of the nose and accessory sinuses as a result of obstruction of the opening of a mucous gland, the so-called cysts found so frequently in the pedunculated growths in the nose are in reality pseudo-cystic in character and are simply dilated connective tissue spaces filled with serum. They are not lined with epithelium as are the true cysts, but with endothelium derived from the connective tissue in which they are formed.

Antral, Choanal or Nasopharyngeal Polypi. The edematoses in general may have their origin from any portion of the mucosa lining the nose or accessory cavities but spring most frequently from the ethmoid cells and middle turbinate. When arising

from the antrum they have attracted enough attention among rhinologists to have been given a special name, that of antral, choanal or nasopharyngeal polypi. Histologically they are identical with the edematoses as described above but their peculiar origin and location make them worthy of special consideration in this connection. Since Zuckerkandl in 1893 called attention to their origin they have been a subject of considerable interest and Woakes, Hajek, St. Clair Thompson and Killian among others have treated the subject thoroughly and have established beyond question the fact of their origin in the antrum. Avellis (4) in 1913 and Ewing (20) in 1918 have reviewed the subject again and Ewing has taken up that portion of the subject which is germane to this paper. He reports thirty-five cases of which fifteen were studied histologically and his observations in this field agree with those of the author (5) expressed in a previously published paper but based on a much smaller number of cases. His conclusions regarding their development differ from mine in one detail, namely, the mode of exit of the polyp from the antrum. It has been my conclusion from a study of the histology and the gross pathology at operation that these growths start as either an edematous fibroma or as a cyst of the mucosa from a chronic inflammatory process, probably not suppurative. This fills the antrum and extrusion through the antral opening occurs as a result of pressure. Plausibility is lent to this theory by the fact that at operation the antrum is usually found to be filled by the growth, that studies of the antral mucosa show an older or more advanced stage of pathological change in the antral tisues and because this is the only reasonable, logical conclusion that can be entertained. Ewing maintains that it certainly does not occur in this manner for the reason that "the antral portion has often been found to be quite small, occupying only a small portion of the posterior part of the cavity." Such is sometimes the case, but since histologic observations show that the antral portion of the growth is always of quite marked fluid content, it is quite logical and reasonable to suppose that after its extrusion through the ostium and its resultant free expansion in the nose there occurs a migration of the fluid in the antral tissues into the nasal portion. This would be favored by the greater fibrosis of the older pathological condition in the antrum and would result in the apparent loss of substance in those tissues. Or, knowing

as we do how rapidly under certain conditions it is possible for edematous tissues to shrink from absorption of their fluid contents, is it not logical to suppose that such a simple occurence might explain the condition in some instances?

Pure Fibroma. Although pure fibroma, commonly called hard fibroma or fibroma durum, is exceedingly rare in the nose and accessory cavities, authentic cases have been reported. My search of the literature has revealed but three; one reported by Meigas (37) as Fibroma of the Septum but with no histology has been eliminated as having probably been an edematous fibroma as no other case of pure fibroma of the septum has been reported. Another, not included among the three, is reported by Dabney (15) as having undergone sudden malignant degeneration which leads to the belief that it was fibro-sarcoma from the beginning as malignant degeneration is questionable in pure fibromata. The original sections in this case may not have reached sarcomatous tissue. Worthington's case (48), however, appears to be authentic as is also that of Dempsey (17) particularly as the latter underwent retrogression and disappeared spontaneously. This growth sprang from the sphenoid and was not limited to the nasal cavities, extending into the nasopharynx as well. Coates case (13) of multiple fibroma had two parts, the nasal part being separate and distinct from the nasopharyngeal, and for that reason it has been included as being a nasal fibroma. Nasopharyngeal fibromata are more common and although outside the scope of this paper their histology is identical.

Here again we meet the same difficulty in the literature resulting from loose application of descriptive terms and in view of the fact that most of the benign growths of the nose are, in a sense, fibromata the adoption of a more accurately descriptive term is suggested, that of "Pure Fibroma".

The histologic structure of pure fibromata consists practically entirely of closely packed fibrous tissue, showing very few nuclei, blood vessels or cellular forms and none of the characteristics of the nasal mucosa. They may be the seat of hyaline change or of calcification. They have their origin in the periosteal connective tissue and Zandonini (55) ascribes an etiological significance to inflammatory processes.

Papillary Fibroma. This subject cannot be properly treated without reference to Wright's (50) admirable review of the subject

wherein he took issue with Hopmann and others and showed beyond question that Virchow's terminology of papillary fibroma should be adopted as the descriptive term for these growths, limiting the term to mean a tumor consisting of a profusely proliferated epithelial layer supported by a very small amount of connective tissue with small, poorly developed blood vessels. The epithelial layer has separating it from the connective tissue a delicate basement membrane, demonstrable in most cases, and a few round cells may be found scattered among its fibres. Mucous glands should be absent from the connective tissue stroma as the tumor develops from the epithelial and subepithelial layers of the mucosa, which contain no glands.

Careless histologic diagnosis has caused great difficulty in the interpretation of the reports of some observers for the reason that they have often been led to make a diagnosis of papillary fibroma by the papillated appearance of the mucosa caused by infolding from pressure and without characteristic epithelial proliferation. It is safe to state, however, that the occurence of papillary fibromata in the nose is rare and their commonest site is on the septum. Arrowsmith (3), in 1911, reported the thirty-fifth case up to that time, not including the one of Blumenthal (6) reported in 1910, all of which were septal growths. Herxheimer (28), in 1911, mentioned one case originating in the frontal sinus, Hannemann (26) one originating in the ethmoid and sphenoid, reported in 1912, Anziolotti (2) one in the same year, Johnston (30) one and Brueggemann two (8) in 1913, Callison (9) one springing from the region of the bulla and middle turbinate in 1916, and Ide (29), in 1917, a case in which two were present, one springing from the septum and one from the anterior end of the inferior turbinate. In all of these we are justified in concluding that the histological diagnosis was carefully and properly made and although all were not located on the septum it is fitting that they should be considered and classed together as papillary fibromata, regardless of their point of origin.

Scarlett (44) calls our attention to another question of interest in this connection, that of the so-called hard and soft varieties of papillary fibromata. This nomenclature has followed the rule that if composed of squamous epithelium the tumor is called hard, if composed of columnar epithelium it is called soft. It is obvious that such a method of terminology is purely arbitrary

and should form no part of the histologic diagnosis. It should be replaced by the terms "squamous celled" and "columnar celled" and in Herxheimer's paper (28) he has made use of these terms, calling his case one of "fibro-epithelioma papillare cylindrocellulare".

Adenoma and Adenofibroma. Here, as in almost all benign nasal growths, one finds in the literature a confusing lack of agreement as to proper terminology. Ewing's observations (19) suggest that the only true non-malignant adenoma is that owing its origin to embryonal tissue, and adenomatous morphology in general is due to various forms of irritation, functional activity or overgrowth. He recognizes, however, that it is not always possible to make the distinction on morphological data alone without considering the conditions under which the process develops and its clinical course. Wright and Smith (52) also state that "benign adenoma unassociated with marked evidence of its origin in the stages of chronic inflammation of the nasal mucosa is an exceedingly rare phenomenon". The most interesting case encountered by the author in this connection is that reported by Gault (21), in 1911, in which the growth is described as filling the whole nasal cavity and attached to the external wall at the ethmoid level and to the roof and necessitating the Moure operation for its removal. The histologic diagnosis of benign adenoma was confirmed by Professors Jacques and Hoche of Nancy and no evidence of recurrence was noted after five months. Caldera (11) in the same year reported a small tumor growing from the inferior turbinate and the meatus the histology of which was that of adenoma. These, with Mosher's case (38) and two of Wright's, are the only ones met with by the author that could be classed as benign adenomata and Mosher, in his article, quotes Hasslauer as reporting but four cases up to 1900. On the other hand, growths partaking of the nature of adenomata but in reality adenofibromata are not so uncommon in the nasal passages. Johnston reports two cases, one in 1909 (31) and one in 1913 (30) which he calls fibroadenomata and describes as consisting of masses of fibrous tissue surrounding islands of glands. Allen (1) also reports such a case which he describes as normal appearing gland tissue embedded in dense fibrous tissue with areas of calcareous degeneration and some areas of new bone formation. Myxomatous degeneration was also evident in places in the fibrous tissue.

It is clear that these latter cases fall into the class of inclusions and cannot be considered as true adenomata, being primarily fibrous hyperplasias which include in their mass islands of normal gland structure. Their proper descriptive term, therefore, should be that of "adenofibroma", thus reserving the term "adenoma" for such rare occurrences of this tumor as may present themselves.

Wright (51), in reporting two cases denominated by him benign adenomata and which were practically identical histologically, gives the following description of their appearance microscopically:"——numerous finger-like processes springing from a common base. These processes are made up of loose edematous fibrous tissue covered by one or two layers of columnar ciliated epithelium; but it is apparent that the epithelial development is a complex one. In no place is there any marked thickening of the layers, but the surface epithelium is seen to communicate and to be continuous with deep indentations and ramifications of it into the underlying stroma. Separate rings of epithelium, ovoid, circular, or ramified in shape, and varied in extent, are seen to occupy and make up a large part of the bulk of the growth. Examined with the high power one is immediately struck with the amount of mitotic granules in the epithelial cells. Evidently proliferation is rapidly going on, but not in such a way as to increase markedly the number of layers." This description is so much superior to any of which the author is capable that he quotes it as a substitute, particularly as it has not been his fortune to see sections from a true nasal adenoma.

Angioma and Angiofibroma. These growths, most frequently termed bleeding polypi of the septum, are also subject to much confusion in their terminology, and for several reasons. Primarily, they were thought to be limited to the septum but Malan (32) in reporting two cases described by him as telangiectatic fibroma and telangiectatic adenomatous fibroma suggests the term bleeding polypi of the nasal fossae. Citelli (12) in reporting five cases, one of which was not situated upon the septum, deals strongly with this part of the subject and advocates discarding the term bleeding polypi of the septum. Matsui (35) reports two cases, both of which he speaks of as bleeding polypi of the inferior turbinate, describing them histologically as consisting of "hyperplasia of connective tissue and increase in vessels, the

latter greatly dilated." The cases of Mayer (36) and Callison (10) were both of septal origin, the former defined as a "cavernous angio-fibroma" and the latter as a "polypoid haemangioma". Reamer (41) reports one case of angioma of the antrum histologically pronounced benign. Thus it may be seen that the terminology should not include the location, for although most such tumors spring from the septum they may have their origin from other parts of the nose and accessory cavities.

This conclusion, however, does not settle the vexed question of terminology and classification as applied to them. Mosher (38), referring to Haslauer's statistics, leaves the impression that the preponderance of benign new growths of the nasal cavities are angiomata, as out of 115 growths reported 57 were of that type. Wright and Smith (53), on the other hand, state that "nasal angioma is a rare form of tumor". This evident disparity is explained by the usual laxity in application of descriptive terms and is probably due to the fact that apparent tumors consisting of simple granulation tissue are comparatively common, especially on the septum. Citelli (12) states: "It will be observed that the etiology of bleeding polypi is the same as that of all tumors, or of any neoformations upon which irritant causes have a marked influence. These irritant causal factors sometimes set up simple or specific granulations, at other times true tumors. These granulations must not be confused with the veritable tumors." Russo (42) also, in reporting five cases under the title of bleeding polypi of the nose, recognizes this differentiation by diagnosing two of his cases histologically as granulation tissue only. He also refers to Citelli's statement to the effect that with the denomination of "bleeding polypi" tumors of various kinds are united in the same class, as for instance, the granulations and pure angiomata; and quotes Mascaigne, Casalis Brunetti and Denogany who seem to think that all so-called bleeding polypi should be united in one group and, taking account of their histological and clinical characteristics, called "fibrovascular benign tumors of the nose". In spite of all this Russo clings to the old term, thinking a change would only confuse and would solve the problem for no one but conceding that among the bleeding polypi there should be included neither the pure angiomata nor the granulation tissue masses.

Citelli, in the paper referred to above, goes on to discuss at some length this part of the subject, proposing that "all nasal

tumors be classified according to their histologic structure, following the actual rules of pathology", and after a detailed description of his case states: "As a matter of fact, the larger part of the nasal tumors to which the designation 'bleeding polypi' has been applied are true angiomas: simple angiomas, cavernoma (or more often mixed, simple and cavernous); or mixed angiomas, with connective or myxomatous tissue." Personally, the author can think of no good, scientific reason why these tumors should not be classified according to their histology as: Granulation tissue masses; Angiofibromata, either simple, cavernous or mixed; and Pure Angiomata, either simple, cavernous or mixed. The term angiomyxomata would be inapplicable here for reasons previously elaborated.

It seems likely, from a study of the description of many of the cases reported by Citelli, Russo and others, that the term "angioma" has in many instances been applied to tumors more properly falling into the present classification of "angiofibromata". There must, of course, be a certain elasticity in the application of these terms and the personal bias and opinion of the observer must be taken into consideration.

Histologically, the granulation tissue masses are simply collections of rapidly growing blood vessels, their walls formed by a single layer of endothelium and supported by a stroma of young connective tissue containing many cells.

The angiofibromata are made up of blood vessels supported and surrounded by fibrous tissue of varying degrees of density and in some instances the site of edematous infiltration. The vessels, particularly those of capillary size, often consist solely of a single layer of endothelium although in many of the older tumors with larger vessels the vessel walls may present both fibrous tissue and muscular structure, the latter rarely. The vessels run indiscriminately through the tumor mass so that in sectioning they are cut at various angles. Endothelial proliferation in the extra-vascular substance is common, particularly in regions showing rapid growth of small vessels, so that in some cases the inter-vascular structure is largely made up of endothelial outgrowths from the vessel walls. These cells sometimes form themselves into rows as if in an effort to form new vessels. The cavernous types usually show the above picture in some sections, with the addition of dilated blood spaces in others. These spaces frequently show an increase of fibrous

tissue in their walls including proliferating endothelial elements and some fibroblasts. Extravasations of blood into the fibrous tissue stroma are frequently observed. The epithelial covering is usually stratafied and may be either the normal columnar or squamous, the latter usually in the anterior part of the nose from local irritation.

The pure angiomata, practically never met with in the nose, are made up practically entirely of vessels with virtually no fibrous supportive structure and may be either capillary, arterial, venous or cavernous. Their intervascular substance may be made up entirely of endothelial elements and their covering is subject to the same variations as above.

Angiomyoma. Citelli (12), in the paper referred to above, adds the report of a case of nasal tumor which he defines as an angiomyoma and describes as consisting of capillaries, mainly arterial, which presented a marked hyperplasia of their muscular layer. These vessels were interwoven with and surrounded by bundles of pliable muscular fibres with characteristic elongated nuclei and such fibres enveloped the surface of the tumor like a capsule. It appears that in this instance there occurred in the muscular coat of the vessels a marked hyperplasia resulting in the above histological findings and as it is the only case of its kind on record is of corresponding interest.

Lymphangioma. This condition is so rare in the nose as to receive no more than passing mention in the literature. Johnston (31) mentions a case reported by A. Schmidt, and Wright and Smith (54) refer to one case reported by Hamm and also to one reported by Hajek and Polyak (27) called lymphangiectasis myxoma but which they suspect was a myxosarcoma.

Histologically, lymphangiomata are tumors composed of lymph vessels instead of blood vessels as in the haemangiomata and their microscopic appearance is very similar to the latter. Never having seen a lymphangioma of the nose, the author will not attempt a description of one.

Papillary Hypertrophy of the Turbinates. This type of hypertrophy is sometimes so marked as to form what is practically a tumor and will be included in this paper for that reason. Although most common on the posterior end of the inferior turbinate, it has also been observed on the anterior end and on the posterior end of the middle turbinate. Its pathological interest is the same, regardless of its site, and one description of its histology will suffice for all.

The chief factor in the histology of this condition is the marked hyperplasia of the fibrous tissue in the sub-epithelial layer, usually unaccompanied by edema. This results, of course in engorgement of venous spaces, in partial obliteration of glands from pressure with dilatation of some of their acini, and in hyperplasia of the epithelial covering. The latter occurs as a direct result of the papillation of the surface produced by fibrous overgrowth, necessitating epithelial proliferation to keep pace with the increase in surface area. Metaplastic changes are also frequently met with in certain places, accounted for by the irritating effects of pressure and disease upon the epithelium.

Chondroma. This type of tumor is also of great rarity in the nose although many such have been described by authors who have doubtless taken septal spurs (ecchondromata) to be such. Sherrill (46) reports a case of ecchondroma of the antrum and Schwerdtfeger (45) also reports a tumor diagnosed as genuine chondroma of nose, frontal sinus, antrum, ethmoid and sphenoid. His histologic findings are quoted: "The section shows the tumor consisting of several small nodules, connected with little tissue containing few blood vessels. The cartilaginous tissue itself contains numerous cartilage cells and some homogenous matrix, negative to elasticin staining and presenting in spots distinctly fibrillary structure." He concludes that these areas show commencing mucous metamorphosis and if this is true we would here have an example of true myxomatous tissue in the nose. He is led to this conclusion by finding spots wherein the matrix separates into fibres, with evidence of mucous degeneration of the protoplasm of the cartilage cells in the more central portions of the growth. Guggenheim (24) reports a case which he considers benign, with photograph and description, and Moure and Pierre-Nadal (39) report a case of osteo-chondroma of the nasal fossae, without histology.

Osteoma. A number of cases of osteoma of the nose and accessory cavities have been reported, probably occurring with greater frequency in the sinuses than in the nasal cavities proper. Marx reports three cases (34), one originating in the anterior ethmoids, one in the frontal and one in the sphenoid. Culbert (14) also reports an osteoma involving multiple structures including the ethmoid and frontal sinuses while Boardman and Donovan (7), reporting a case of osteoma of the frontal sinus cite Sewell's paper wherein he states that he found ninety-two cases of osteoma frontalis in the literature. Thus, it may be seen

that osteomata are not uncommon, but they must not be confused with bony occlusions of the posterior nares such as that reported by Rutland (43) which are developmental defects and not new growths. Also, they must be differentiated from exostoses, frequent in the nasal cavities, especially on the septum. Histologically, they usually consist of dense lamellated bone with few Haversian canals although they may consist of spongy bone with many vessels and abundant marrow spaces and cells.

Lipoma. Goldstein (23) reports the only case of lipoma of the nose or accessory cavities which the author has been able to discover in the literature. His case is described as being a fatty tumor mass in the antrum, the histology of which was reported by Thompson to be as follows: "Connective tissue stroma, holding fat cells in its meshes and enclosed in what appeared to be a fibrous capsule. Small capillaries were found in the mass and the cells were typical of fat cells of the adult type, although some were larger than those usually found in fatty tissue."

Teratoids. Of this rare condition, a true developmental phenomenon, but few cases appear in the literature and but two with histological description have come to the author's notice. Onodi (40) describes his case as consisting of stratified squamous epithelium, hornified, papillated and containing hair follicles and sweat and mucous glands. The stroma was formed of hyaline connective tissue, containing fibroblasts. Haenisch (25), in 1916, gave virtually the same description of his case except that it contained muscle, mucous and serous glands and adipose tissue. In addition, Gaudier (22) reports a case of cyst of the middle turbinate of dermoid contents and concludes that it was probably congenital. These tumors are not teratomata, being too simple in their construction for that term to be properly applied to them but the first two evidently fall into the classification of nasal teratoids, the latter being, of course, a dermoid. They are probably of little more than passing interest as their occurrence is so rare that no one individual is likely ever to encounter one in practice.

Conclusion. From the foregoing it would appear that our classification of nasal growths should be revised and the revision adopted by rhinologists as a whole in order to avoid the confusion resulting from the loose application of terms with indefinite meaning. Thus, we should be enabled to differentiate nasal neoplasms according to their histogenetic and pathological characteristics and not according to their morphology as

we are now constrained to do. First, of course, we have to contend with the question very general among pathologists as to what constitutes a true neoplasm and whether any of the benign growths in the nose are really neoplastic when histogenetically considered, for so close a relationship exists between the products of inflammation and new growths in the nose that it is at present too difficult a task to differentiate them. It would seem, however, that for practical purposes as rhinologists a classification accepting nasal growths as they are and based on their genetic and histologic characteristics would be sufficient and certainly helpful.

And finally, although many of the conditions mentioned herein are rare, it is the opinion of the author that if more rhinologists had a slight acquaintance with the histopathology of the nose the number of reported cases would be greatly increased. It is not always sufficient to refer specimens to a clinical pathologist, for his knowledge of gross nasal pathology may be insufficient to aid him in the recognition of conditions which, while quite common in general pathological diagnosis, might be rare and interesting specimens when considered in relation to their origin and causation.

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LATENT EMPYEMA OF THE MAXILLARY ANTRUM.

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The significance of localized pain and unilateral purulent discharge, with an increase of both when the head is lowered, were recognized as indications of antrum disease long before the writer began special practice. After two years of intensive training and five years of practice in a fairly good clinic he felt that, with the aid of head mirror and probe, transillumination and roentgenograms, he was able to diagnose nearly any case of empyema of the antrum. Any remaining doubt was to be definitely settled by irrigation of the cavity, however, several times during the past few years he has had some surprising and humiliating experiences, several of which will be given you in detail. To some of the Fellows this paper will seem elementary and semi-scientific because it is unaccompanied by bacteriologic studies. However, let us hope that a clinical report is not altogether useless. Most of the cases seemed unique at the time they were first recognized; and had the writer known of similar conditions in the practice of other men, he would have been spared no little chagrin and embarrassment. All of these cases were first thoroughly studied by an internist who had made general physical examinations including urinalysis and Wassermann test. When all the examinations had been completed they were referred to me for examination of the eye, nose, ear and throat for the detection of some condition that might explain the chief cause of complaint.

Case Reports.

CASE No. 1. Mr. K., White male, 38, a laborer at a saw mill was referred by Dr. J. S. McLester for *frontal headache*. This symptom was present all day and sometimes in the night but seemed to be increased by physical labor. The ears, nose and throat were normal so far as one could tell by inspection. Transillumination was negative. Roentgenograms were negative. The man had 1.25 diopters of far sight and was given lenses with the advice that he wear them constantly, and it was explained to his physician that this was the only condition recognized that might account for the headache. Three weeks later the man reported "no improvement" in his condition. He was given provocative doses of mercury, and a second Wassermann test made by his internist was likewise negative. Two weeks later while driving a wild mule he was thrown from a buggy against a sharp projecting rail of a worm fence.

Sometime after this accident a friend brought me this message:—"Kelly said tell your doctors in Birmingham that a rail splinter let out an ounce of corruption from his face and cured his headache; and he has come to believe that the old son-of-a-Jackass had more sense than all of the Birmingham doctors."

It is possible that an irrigation of this antrum might have revealed the trouble but all other tests were negative and there was nothing to suggest the need for irrigation.

CASE No. 2. Mr. B., White male, 33, was referred by Dr. Jno. T. Kent because of *acute severe pain in left malar region.* There was a free mucopurulent discharge from middle left meatus, increased by lowering the head; and one immediately made a clinical diagnosis of acute antritis. The diagnosis seemed fairly clear and the antrum was irrigated with the removal of several cubic centimeters of pus. Owing to the narrowness of the normal drainage channel into the nose, the procedure was so extremely painful that the patient absolutely refused to have the irrigation repeated under a local anesthetic. He insisted upon general anesthesia with an opening for free drainage. He urged that I make a similar opening into the antrum on the other side but it was explained that this was useless as he suffered no present pain and no discharge. Nevertheless he argued that several years ago he had experienced pain on the right side and felt that there might be trouble there in the future. He was so insistent that his family physician promised to stand by and see that the right antrum was thoroughly irrigated. Preferring to operate upon the clean side first, we inserted a trocar into the right antrum and were much surprised at the removal of one or more cubic centimeters of thick greenish pus. It was believed that this was probably of a few days duration and had resulted from the same acute coryza that initiated the acute empyema of the left side. His physician insisted that I make a large opening through the canine fossa so that this cavity could be inspected and it then developed that the right antrum, formerly free of pain and discharge, contained three or four polyps—half filling the cavity. So far as I could ascertain, the condition had no connection with the teeth. It is possible that the transillumination and the roentgenograms in this case were both negative because the diseased condition was bilateral. As in the case first mentioned, the trouble on the right side had never been suspected and was discovered almost as by accident.

CASE No. 3. Miss S., White female, 27, complained of *headache and face ache between and below eyes off and on for ten years.* This lady had had six operations by four different specialists before consulting me, and each time had received marked, though temporary, relief. The anterior half of each middle turbinate had been removed, the sphenoids drained, each frontal sinus had been opened externally, and it was easy to pass a probe through the nose into these frontal cavities. She came demanding an operation upon each antrum, and her frequent use of the term "Pan-Sinusitis" showed her familiarity with nasal surgery. At this visit she was carefully examined but there was no symptom other than pain, and there was no sign suggesting antral disease. Transillumination and roentgenograms were negative. She had an error of refraction which had not been corrected, and she was given lenses with the request to return in three weeks if she had not greatly improved. She came back with the usual complaint and this time each antrum was irrigated though it hardly seemed indicated. From the left side a little

pus was removed and the water from the right side returned unchanged. The pain was unrelieved by repetitions of this procedure; and some days later under ether the left antrum was freely opened and found to be literally packed with polyps. They were flattened with compression.

The right antrum was not opened. The patient got relief for several months but returned in about a year complaining as before, and demanded an operation upon the other antrum. The opening of the left antrum into the inferior meatus was still patent and a third set of radiograms showed no recognizable change since the operation upon the left side. Transillumination was still negative. Pain between the eyes was unbearable. The ethmoid and sphenoid cells were apparently open, and the patient urged operation upon the right side. After consultation with a rhinologist who had operated upon her on a previous occasion, I reluctantly consented to open the right antrum, explaining to the patient that relief was very doubtful. A large opening in the canine fossa revealed a most unexpected condition. The cavity in the antrum was very small, the internal and superior walls were lined with velvety mucous membrane apparenty normal. Toward the posterior angle and filling the external half of the antral cavity there was a dark colored mass with a rather dull glistening sheen which was at first thought to be mucous membrane. It was easily indented by firm pressure and did not bleed on gentle curetting. It proved to be cholesteatomatous material (?) and on removal presented the layer-like formation seen in the onion. The underlying bone was bare of all mucous membrane and in the extreme posterior and inferior angle we found a small osseous cyst of six or eight millimeters in diameter connecting with the antral cavity by a small hiatus that admitted a two millimeter probe. This bony wall was crushed with a curette and on the inside was found a coil of hair—six strands—from eight to twelve centimeters long and a hard tooth-like body, two or three millimeters in diameter, that appeared to be dentine. The anesthetic was given by Dr. W. B. Hardy who was then preparing for work as a rhinologist. I have been unable to find the record of a dermoid cyst removed from the antrum. However, I have not exhaustively searched the literature. In view of finding this tooth-like structure we went back and carefully studied the radiograms expecting to find a shadow suggesting this bony cyst and its tooth-like secret, but all was negative. This patient had a fulminating appendicitis on the fourth day but survived the operation and was greatly improved for a year. The headaches then returned and finding myself unable to determine any cause for the symptoms, she was sent to Dr. H. P. Mosher, of Boston, who kept her under observation for several weeks, went into every accessory sinus, could find no cause for her suffering, and wrote me very frankly that he could not help her though he believed her suffering was very real. Sometime later she fell into the hands of an ambitious surgeon who opened her skull and said he removed a part of the Gasserian ganglion. She survived the operation and lived a year but was never free of pain. I saw her no more until a few days before her death when I was asked to visit her at her home. This time she was unconscious, with unequal and dilated pupils, stiff neck, cloudy spinal fluid, and she died with all the symptoms of meningitis, probably resulting from some undiscovered ethmoid cell. I was unable to secure an autopsy. The poor girl was a martyr to the limitations of our specialty.

Just ten years ago Dr. Haskin, our Secretary, read a paper

before this Society, then meeting in Washington as today, in which he called attention to apical abscesses as a frequent cause of disease. He was convincing, and at the same time so surprising, that I spent the following summer in Boston learning what I could about the relationship of teeth to rhinology and to general infection. Returning home in the fall I convinced a dental friend of mine of the importance of studying cases together and since that time we have had a joint reception room and with his assistance and the valuable data suggested by his excellent roentgenograms, have been constantly amazed at the importance of diseased teeth and of latent osteomyelitis of the alveolar process from which teeth had been removed, sometimes as long as fifteen years previously. The remaining cases reported have direct association with dental disease.

CASE No. 4. Mrs. W., White female, 43, was referred for an opinion as to a possible focus of infection that might produce *extreme physical fatique*. The general history was negative except that several years previously she had suffered similar symptoms and had secured marked (though not complete) relief from the removal of badly diseased tonsils. She was an extremely fleshy woman. Her condition had been thoroughly studied by an internist who could find no cause for trouble. The ears, nose and throat were free of all symptoms or signs of focal infection. Roentgenograms of the teeth were negative except a suggestion of a possible granuloma in the right upper jaw from which a molar tooth had been removed several years previously. The dentist suggested the wisdom of opening this bone and curetting the area, explaining, however, that he was not certain of the presence of any disease. Remembering my former chagrin I decided to irrigate both antra of this patient, to be sure of excluding empyema. Roentgenograms and transillumination having already been negative the irrigation was likewise negative. It was finally decided that the dentist should open the bone and cut the suspicious area. The bone proved to be rather soft, more or less cancellated with small white deposits of pus which seemed to increase in number and in size as one approached the antral cavity. The dentist remarked to me that he believed that it would extend into the antrum but he was urged to continue wherever the bone seemed necrotic. Finally he broke into a cavity containing one or two cubic centimeters of thick, brownish gray, purulent material, and it developed that it was within the antral cavity but covered by the unopened mucous membrane that had formally been attached to the floor of the antrum. This membrane was now elevated just as we elevate the mucous membrane for a sub-mucous septum operation. The dentist had produced perfect anesthesia, by blocking the nerves, and on his urgent advice I proceeded to open the canine fossa, explaining to the patient that such extensive detachment of mucous membrane might mean further disease within the antrum notwithstanding the negative results of our irrigation. Several polyps were removed and a good deal of diseased mucous membrane. The patient remained well for six months and returned with the usual symptom of excessive morning fatigue with nothing whatever in the roentgenograms that I could detect, still

the dentist said he felt there was an abnormal appearance of the bone in the left alveolar process. A tooth had been removed many years previously and a small root had been left. The dentist undertook to remove this root with the indefinite hope that he might find some focus of infection. A large flap was turned upward and immediately upon entering the bone he discovered soft cancellated material as had been detected on the other side six months earlier, and the deeper he went, the softer the bone appeared to be following the channel of the old tooth root that had been removed twelve years previously. The condition extended right on into the antrum but not through the mucous membrane. The intact mucous membrane explained why the irrigations had always been negative. The antral surface of the mucous membrane was edematous and leathery; and within the cavity we found a number of small polyps and a little pus.

CASE No. 5. Mr. C., White male, 24, was referred by Dr. Pugh for *rapidly failing vision in the right eye*. The man had an acute uveitis with fixed pupil and cloudy vitreous. An internist excluded as far as possible any systemic disease, especially could he find no evidence of tuberculosis, or diabetes or lues. There were no symptoms or signs of accessory sinus disease. As a special precaution each antrum was irrigated. Roentgenograms showed no evidence of apical abscesses, but about the root of one molar there was a "pus pocket" extending far down alongside the root of the tooth. The tonsils were very badly diseased and it was decided to remove them at once and have his teeth attended to afterwards. About eight days after his tonsillectomy the dentist proceeded to clean up the suspicious area at the root of the second molar tooth. He was surprised that it had extended far beyond the area suggested by the roentgenograms and remarked that he was afraid it was another case of "mastoid disease" of the upper jaw. (I had frequently referred to the fact that the necrosis that he had heretofore found was very similar to the cancellated tissue noticeable in certain forms of mastoid disease, differing, however, in one particular, namely, that in a long-continued chronic mastoid the bone is often hard while these long-continued inflammatory areas in the alveolar process suggest the conditions found in the mastoid cells in the more acute cases). It became necessary to remove the tooth because of the extensive disease around its roots and these apparently did not enter the antrum. In curetting the cavity, however, the dental curet easily entered the antral floor and there was a discharge of a few drops of pus. Influenced by some former experiences we thought it wise to inspect the antrum, and made a large opening through the canine fossa finding very little pus, but four large polyps one of which almost filled the cavity and all of them springing from the inferior wall.

Within the last four years we have found seven other very similar cases where the antrum has appeared to be involved from chronic infection from long standing disease of the alveolar process from which the teeth had been removed years ago. Two were being investigaatd for optic nerve disease, one for chorea, one for unexplained elevation of temperature, two for kidney and ureteral disease and one for chronic arthritis.

It should be mentioned that in the earlier cases there had been no curetting of the root sockets as is now practiced in the extraction of dead teeth.

Conclusions.

(1) The maxillary antra may become infected from diseased alveolar processes from which teeth have been removed years previously. In all cases so far recognized there have been small amount of pus and many polyps.

(2) Latent purulent infection of the antra may exist unsuspected and produce serious damage to eyes and other organs.

(3) In bilateral disease roentgenograms and transillumination are often negative and these measures may fail us even in unilateral cases.

(4) The bony antrum may harbor a chronic abscess that lies under the mucous membrane of the floor—when the latter appears to have a macroscopic opening—permitting the pus to enter the cavity proper.

(5) Negative irrigation is not conclusive proof excluding antral disease.

(6) In chronic empyema of low grade, patients may suffer any form of disease ever produced by virulent focal infection of any other kind.

(7) Unless we devise some form of instrument with periscopic mirror (like the Brown-Buerger cystoscope) for inspection and study of the walls of the maxillary antrum we shall probably soon be driven to exploratory operation, just as abdominal surgeons have long done to clear up mooted points of diagnosis.

DISCUSSION.

Dr. J. E. Mackenty, New York City, said that this was a most interesting series of cases of antral disease. It emphasized the importance of inspecting the antrum in cases which do not clear up under intranasal treatment. The question was whether in the presence of these toxic manifestations, one should open the antrum and run a chance of killing sound teeth. Dentists should have a copy of this paper, as they often show reprehensible carelessness in leaving the bone without curetting after removing diseased roots. The speaker cited one such case in the history of an old sea captain, suffering pain from a necrotic area in the floor of the antrum; curetage of this area removed the pain. The writer personally had suffered from antral disease after removal of a bad tooth. He had temperature hemolysis and hemolytic jaundice and symptoms suggestive of tuberculosis. This all cleared up after treatment of the antrum. The X-ray revealed the condition, nevertheless the X-ray is often negative in antral disease. The antral washings are often not pus, but mucilaginous soapy material, indicating toxicity of the case. The antrum must be widely opened in order to get well.

Dr. J. M. Ingersoll, Cleveland, O., said that the difficulties were often very great in making a diagnosis of antral disease. When the two antra were infected they would appear alike in the X-ray. Sometimes the X-ray afforded considerable help; sometimes not much. The diagnosis must be made by careful exclusion of one factor after another

and study of the problem. Experimental puncture helps very much if there is fluid present. The speaker said that after putting the trocar into the maxillary sinus, he was accustomed to pass in a small silver probe, which would discover some indication of the condition of the mucous membrane. If this does not adhere closely to the bone it confirms the suspicion that the membrane is thickened and helps the diagnosis in doubtful cases. The speaker advocated exploration of the sinus through the canine fossa after the exclusion of possibility of other sources of infection.

Dr. L. E. White, of Boston, said it was interesting to know of some source of infection going on in the body, which might remain unknown. Some of these cases might be due to infected alveolar process, and washing out of the antrum would do no good. Irrigation does not exclude antrum disease in all cases. Where transillumination shows a difference in the two antra, there is probably disease. In one patient there was a swelling on the floor of the nose. He also suffered neuritis and arthritis. The tonsils had been removed, but the second bicuspid was a pivot tooth. The dentist did not find any particular marks of infection, but after removal of the old root the symptoms cleared up.

Dr. R. B. Canfield, Ann Arbor, Mich., said that in some cases a denticyst might cause infection, by breaking down and becoming infected. The Hays nasopharyngoscope was a useful instrument. Treatment of nasal-accessory-sinus disease is unsatisfactory; sometimes they get well; sometimes they don't. Making a large opening in the nose is important, but they get filled up when they have a cold. Irrigation can be done through an opening in the canine fossa, made under cocain. In some cases a collection of edematous mucous membrane fills up the opening.

Dr. Harris P. Mosher, Boston, Mass., said that he had learned two things at this meeting: one yesterday from Dr. Dean; one from this paper. The speaker thought that the question of opening the antrum through the canine fossa should be settled with regard to the possibility of killing teeth. He was in favor of opening the antrum widely. West of the Mississippi they were afraid to do this, as people had brought law suits. Clinically this has not been proved, and the matter should be decided.

Dr. F. P. Emerson, Boston, Mass., said that there were two points in reference to diagnosis. The X-ray value depended on whether the pathology started in the ethmoid or the jaw. In the ethmoid, one should use Waters' position. The most difficult cases are where there is no pus, but atrophy with accumulation of polyps. In infections from teeth, the pathology is all on the floor of the antrum. In other cases it is opposite the middle turbinate. Dr. Emerson emphasized what Dr. Mosher had said. He had worked with a dentist who assured him that the vascular supply of the teeth was excellent, and not affected by opening the antrum.

Dr. Wendell C. Phillips, New York City, said that antral disease was more common than generally supposed, but one should not go away with the idea that a diseased antrum produced general infection every time. General infection is extremely rare. In the case of a man who took his wife to Battle Creek for treatment, he had his own X-ray taken and showed a dark antrum. The X-ray was tried before and after washing the antrum. Air was put in to blow it out, and a polyp was blown out. The X-ray was the same before and after.

Dr. F. M. Law, New York City, said that he would suggest better co-operation between the X-ray man and the surgeon. A discussion between the two would do much to bring out the interpretation of the finer details of the case. Both sides would learn more. Another point: when both antra are involved, they look alike under the X-ray.

A SEPTIC TYPE OF TEMPERATURE NOT REFERABLE TO THE EAR IN CASES OF ACUTE SUPPURATIVE OTITIS MEDIA.

By HAROLD I. LILLIE, M. D., Rochester, Minnesota.

If a patient with acute suppurative otitis media has a septic type of temperature, the natural tendency for the otolaryngologist is to assign the cause of the fever to extension of the infection from the ear and mastoid to the sigmoid and lateral sinuses. It has been well established in such cases that in taking time to make a differential diagnosis, the physician does not endanger the patient. Such a course may reveal involvement of other structures to account for the clinical picture and, thus, danger to the patient and embarrassment to the physician may be avoided. It is not assumed that some absorption may not take place from the ear, but it may be assumed, from the clinical course of the case, that the ear has not been the important factor.

In the five cases reported herein it is believed that four disease conditions acted as causal factors in the septic type of temperature. It will be noticed that all the patients were young children, in whom the clinical course for any diseased process may not be expected to run true to type, thus making diagnostic possibilities difficult.

Case 1 (A358689) Acute suppurative otitis media, pyelitis.—Marguerite A., aged nine months, was examined in the Section on Pediatrics of the Clinic May, 1921 because she had not been well for about a week. The mother had noticed that the child had a high fever every afternoon and was very restless at night. She had lost 5.5 pounds. The stools had been green. Cough had not been noticed.

Examination revealed a temperature of 102.5° and a suppurative otitis media. The leukocytes numbered 41,000. Microscopic examination of the urine showed alkaline reaction, many pus cells, and on culture, *bacillus coli*. All symptoms disappeared under appropriate treatment for the pyelitis. The condition of the ear disappeared after paracentesis and the usual treatment.

Case 2 (A335868) Suppurative otitis media, central pneumonia.—Alfred M., aged thirteen months, was first seen in the Ear, Nose and Throat Section of the Clinic in September, 1920 when a suppurative otitis media was found in both ears. In November, 1920, the child was re-examined and trouble again found in both ears. He was kept under observation in the out-patient service. The records show that the ears would remain dry for one week but without

closure of the perforation, and the next week a discharge might be present. Early in January, 1921, the ears were almost normal.

January 14, 1921, the patient was brought to the Clinic because he had not slept well during the night and had often put his hand to his right ear.

Examination revealed the tonsils to be infected, the nose and left ear normal, and a dry perforation in the right ear, the membrane being milky white in color. The child's temperature was not taken, but two days later it rose rapidly to 104°, and a mucopurulent discharge appeared in the right ear. The child was placed in the hospital for observation. During the night, the temperature dropped to 99°. The possibility of infection of the lateral or sigmoid sinus was thought of, but the presence of a slight cough made an investigation of the chest advisable. The examination was negative, January 20, the leucocyte count was 24,000; the examination of the urine was negative, and culture of the blood was negative. Roentgenograms of the chest showed a definite shadow. The child was treated for the pneumonia. The condition of the ear did not change materially. After twenty-two days the child was dismissed from observation. (Fig. 1).

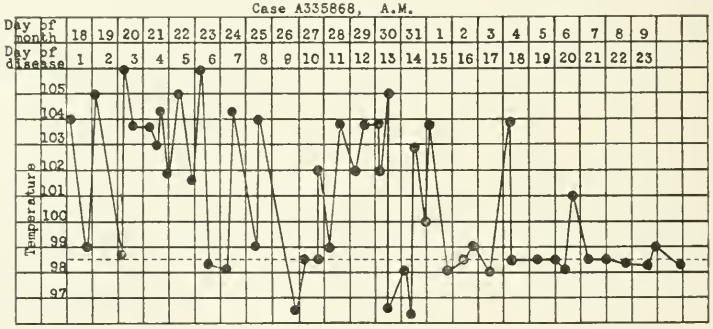


Fig. 1.

Case 3(A348892) Acute suppurative otitis media, abdominal post-operative condition.—Marcella K., aged four years, was brought to the Clinic February, 1921 because of a biliary fistula, following an abdominal operation elsewhere.

The child's temperature was 99°. The biliary fistula was draining. The cervical glands on both sides were enlarged, the tonsils were slightly reddened, the nose and ears were normal, and the chest was normal. Examination of the urine was negative. The leucocytes numbered 6,000; the differential was negative. A culture of the discharge from the biliary fistula showed streptococus and *bacillus coli*. In a few days the temperature rose to 103°, the patient complained of abdominal distress, and developed acute bronchitis and suppurative otitis media on both sides. The leucocytes then numbered 8,900; the differential was negative, and culture of the blood was negative. The increased temperature with chilly sensations indicated sepsis. Examination of the blood for Plasmodium malariae was negative; typhoid fever was ruled out. The condition of the ear appeared satisfactory. The chest remained negative. Further investigation elicited the fact that the child had had attacks similar

to the present illness with chilly sensations and fever. No real causal factor was ascertained except the fistula in the abdomen. The child was dismissed from observation against the physician's advice after twenty-six days. Subsequent correspondence with the mother revealed that a chiropractor was treating the child and that the biliary fistula had closed after two weeks. She had increased temperature occasionally (Fig. 2). A definite diagnosis was not made in this case.

Case 4 (A349322) Suppurative otitis media, gastro-intestinal disturbance.—Theodore B., aged twenty-two months was brought to the Clinic in February, 1921 because he had not talked, because he was extremely restless at night, and had a "head cold". He had had whooping cough at the age of ten months.

Examination revealed definite evidence of rickets and an upper respiratory infection; the temperature was 100°. Laboratory examinations were negative.

March 7, the child was referred to the Ear, Nose and Throat Section because of earache. Suppurative otitis media of both ears was found and paracentesis tympani performed. The child was not hospitalized because of the objections of the parents. There was an afternoon rise in temperature. All laboratory

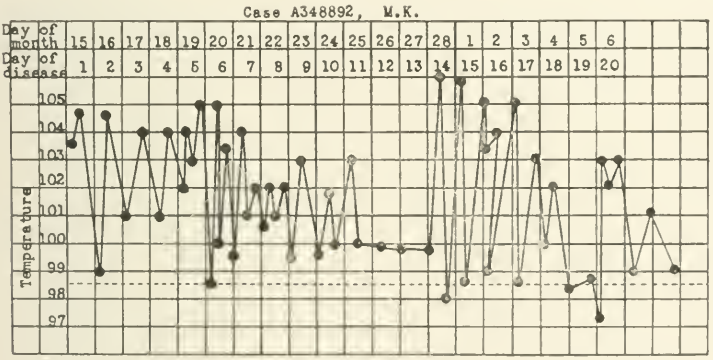


Fig. 2.

and physical examinations were negative. The parents were having difficulty in feeding the child and his bowels did not act normally. Castor oil was given and the temperature became normal and remained so.

Case 5 (A315116) Suppurative subacute otitis media, pyelocystitis.—Alice P., aged six years, was brought to the Clinic in May, 1920, because of a discharging left ear and fever in the afternoons. The trouble had started four weeks before with measles. There had been no chills. While the temperature was down the child appeared to feel well, although the general malaise was evident.

Infected tonsils and adenoids with enlargement of the cervical glands were found. The right ear appeared normal. The left canal was filled with purulent discharge. The membrane was full; the landmarks were not discernible. The discharge was pulsating through a posterior inferior perforation. There appeared to be no drooping of the superior canal wall and tenderness of the mastoid was not elicited.

Many moist râles were heard throughout the chest. The temperature was 99°. The leucocytes numbered 27,000, the differential count was negative. The

urine contained many pus cells. The patient was sent into the hospital for observation and treatment was directed to the condition of the ear and the pyelocystitis. As the urine cleared the child's temperature and general appearance improved. The condition of the ear cleared up under local measures.

Summary.

It is apparent that in this group of children four clinical causes other than the disease of the ear were accountable for the clinical course. At least, treatment directed to the conditions appeared to clear up the symptoms. The deeply situated central bronchopneumonia in Case 2, was the most difficult to diagnose. Operation in this case no doubt would have resulted fatally. The hardly discernible cough was the symptom which lead to the search of the chest for the cause of the symptoms.

The differential diagnosis in Case 3, was established on the fact that the chills and fever, appeared shortly before the onset of the trouble in the ear, and because of the infectious process in the abdomen, although previously there had been no chills and fever from this source.

Pyelitis, or cystitis, particularly in little girls, may be overlooked unless a microscopic examination of the urine is made. Helmholz (personal communication) says that it occurs with upper respiratory infections and that a diagnosis cannot be made unless a catheterized specimen of urine has been examined. He believes that excoriation and redness of the vulva and acid reaction of the urine are not the cause of the rise of temperature, but the result of the temperature. It has been our experience, however, that alkalizing the urine with potassium citrate has caused these symptoms and signs to disappear (Case 5).

Gastro-intestinal disturbances in young children often are the cause of a rise in temperature, particularly if a change in diet has been necessary because of a change in location. With other causes not discernible a brisk cathartic, for example castor oil, is sometimes effective. This procedure is not looked on with favor by many pediatrists.

DISCUSSION.

Dr. Norval H. Pierce, Chicago, Ill., said that he could recall 3 pathological conditions in this connection: malaria, erysipelas, tonsillar infection. These would be divided into two groups, pre-operative and post operative. The first case was one of acute otitis media complicated by erysipelas. The next case was in a man from South America, in whom search for plasmodia was made, because of repeated chills and fever. When the blood was examined at the peak of the temperature, the organisms were discovered. Both pneumonia and tonsillitis may cause these rises in temperature. Another closely related subject is meningitis. In France a nurse with acute otitis media had developed a case of epidemic meningitis, and she recovered with serum treatment. It would seem in these cases it is much better to err on the side of deliberation than to be too hasty in assuming sinus thrombosis. If the patient has sinus thrombosis or meningitis he is doomed in any case. Operation would not save these patients, so it is well to wait a few hours in order to make sure of the diagnosis.

Dr. J. F. Culp, Harrisburg, Pa., said that he had seen a case of a child with double mastoid. The child took a bad cold and had pain and discharge in both ears, temperature 102. One morning the temperature was 106, both ears discharging. Next day the temperature fell to 100. The discharge from the ears stopped. Leucocytes were 18,000, polys 72 per cent. The question was, was it sinus thrombosis? The child was very ill. On the 6th day the internist found a spot on the right lung showing revolution. The child began to cough, and 8 days later was fairly well.

Dr. George MacKenzie, Philadelphia, Pa., said that he wanted to prevent a wrong impression. There had been more mistakes made in not operating on the sinus, and leaving it alone in the presence of thrombosis, than in too many operations. In Politzer's clinic 85 per cent of the cases were fatal where there was a pyogenic infection of the meninges, not operated upon. It is safer to operate than to leave cases alone.

Dr. Lillie (closing) said that it was Dr. Pierce's published conservatism led him to look out for these cases. In pyelitis cases, microscopic examination should be made of the urine. The difficulty of diagnosis of central pneumonia in children, is even greater when otitis media complicates the picture. There is no harm in waiting 48 hours till other causes can be ruled out. There are a large number of spontaneous recoveries from thrombosis of the sigmoid. When the diagnosis is made, operation is easy.

AUDIOMETRIC METHODS AND THEIR APPLICATIONS.

By E. P. FOWLER, M. D., New York City, and MR. R. L. WEGEL, Research Laboratory of the American Telephone & Telegraph Co., and Western Electric Co.

Part 1. Audiometric Methods.

1. *Introduction.*

For a system of mensuration of audition to be satisfactory, it must include measurements of those quanities which are most directly and simply correlated with the condition of its function as a sense and with its pathological condition. The principal function of the auditory sense is to detect sounds of various kinds and wave shapes, varying over a range of pulsating or sound pressure on the ear drum of roughly from about one millionth of a gram to one gram, and over a considerable part of this range to differentiate with certainty between complex sounds so nearly alike that no existing physical apparatus can separate them. Much pioneer work has been done in audiometry by Dr. Seashore of the University of Iowa, and in fact it is to Dr. Seashore that we owe the name "audiometer". During the last few years, progress has been made in the practical application of audiometric methods to otology by Drs. Dean and Bunch of Iowa and more recently by Drs. Wilson and Minton of Northwestern and Chicago Universities.

Considerable difficulty seems to be encountered in the interpretation of audiograms. The purpose of this part of the paper is to report on the results of an investigation into the amount of responsibility for uncertainty of interpretation chargeable to methods and technique of audiometric measurements and to propose remedies. A casual examination will show that careful attention to certain details of methods and technique are essential if a serviceable correlation is to be expected between audiograms and lesions of the ear.

Summary of Part I.

1. Introduction.
   The problem and history.
2. Audiograms,—defined.
3. Frequency scales (2 kinds).
4. Kinds of intensity scales (2 kinds).

5. Importance of proper intensity scale.
   General:
   (a) Importance of parts in use.
   (b) Accuracy of measurement.
   (c) Dynamics.
6. The unit of sensation.
7. Minimum Audibility for pure tones.
8. Maximum Audibility for pure tones.
9. Upper and Lower frequency limits.
10. Area of sensation.
11. Accuracy of the determination of the normal ear curve.
12. Useful audiogram scales.
13. Quality areas.
14. Bone conduction.
15. The Audiometer.
    General:
    (a) The oscillator.
    (b) The attenuator.
    (c) The telephone receiver.
16. Conclusions.

SUMMARY OF PART II.

Application and Conclusions.

2. *Audiograms.*

An audiogram is a curve plotted so as to show the variation of minimum audible sensitivity with frequency where the stimulus is a pure tone. The scales in common use vary considerably both in the spacing of intervals of frequency and of intervals of sensitivity, and consequently in their utility.

3. *Frequency Scales.*

There are two commonly used frequency scales; one in which equal intervals on the chart scale correspond to equal arithmetic intervals of frequency and the other in which equal intervals on the scale correspond to equal logarithmic intervals of frequency. The musical scale is an example of the latter. It is important that some uniform scale of frequency, such as one of those already mentioned to be adopted; otherwise the resulting audiogram curve consists of a number of parts, the shapes of which bear no simple relation to each other and tend to decrease the serviceability of the curve. Since the sensation of pitch change corresponds more nearly to musical intervals than to equal frequency intervals, the musical scale is the more logical to use in an audiogram.

4. *Kinds of Intensity Scales.*

There are two kinds of intensity scales in common use; those in which the sensitivity is given in terms of the stimulus, such as those in which energy or sound pressure is used, and scales of sensation which according to our best information is proportional to the logarithm of the stimulus. Other scales which do not pretend to be either stimulus or sensation scales are sometimes used.

5. *Importance of a Proper Intensity Scale.*

In the study of an audiogram curve, it is natural to stress its peaks or hollows or other characteristics more or less in proportion to their relative magnitudes on the chart. Whether this procedure is justified or not depends among other things on the scale on which the curve is plotted. If two audiograms of the same ear are plotted, one on a scale of energy stimulus and the other on a scale of sensation (log-stimulus), for example, they will in general bear little resemblance. This is particularly true when there are large variations in sensitivity with frequency.

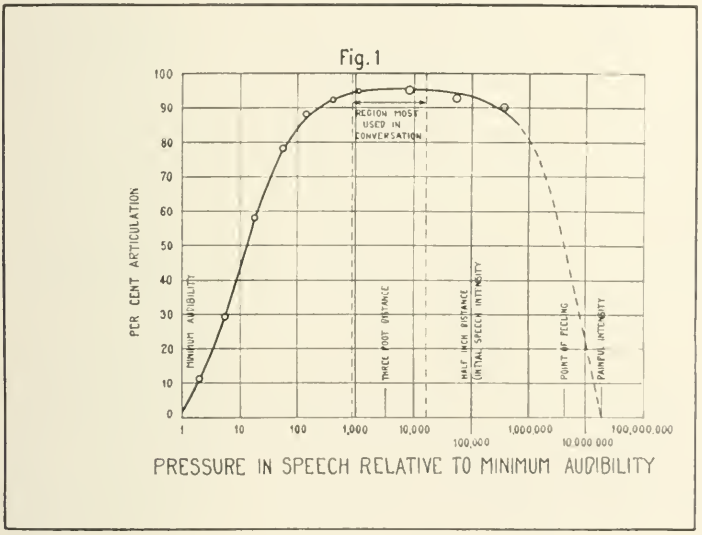
Large undulations in the lower sensitivity parts of the sensation curve will not appear in the stimulus curve and large variations in the stimulus curve will appear on the sensation curve but not as large in proportion. It is evident therefore that the choice of a proper scale is important and it is conceivable that some scales may be of practically no use. There are perhaps three important criteria from which to examine any audiogram scale. They are (a) importance of various parts of the scale in use (b) accuracy of measurement in the different parts of the scale and (c) relation to dynamical characteristics of the ear.

(a) Figure 1 shows a plot of the percentage of meaningless syllables (articulation percentage) interpreted correctly by normal ears over an experimental high quality telephone circuit as the sound pressure is varied over a range corresponding to a ratio of more than 1:1,000,000 (energy flow ratio of a million million).

The unit of the horizontal scale is the common logarithm of the pressure. The pressure at minimum audibility is taken as unity. This curve was given and discussed by Dr. H. Fletcher at the Franklin Institute on March 29, 1922. The plotted points represent 80,000 observations. We will take this articulation percentage as indicative of the importance of the various sound pressures as indicated on the horizontal axis. At minimum

audibility of course, the "articulation" is zero. As the intensity or loudness of a sound is increased, the "articulation" increases to a maximum of 96% at about ten thousand times the minimum audible pressure. This corresponds to ordinary talking at a distance of about two feet from the ear. For louder volumes the articulation decreases approaching zero presumably at a loudness which is acutely painful as shown by the dashed portion of the curve.

If the plot of articulation were made on a stimulus scale on sheets of the usual sixe (8" x 10") it would take six sheets with different scales to plot in energy or three sheets on a pressure



basis. Such plots only cover a range satisfactorily where the largest reading is less than one hundred times the smallest. We must conclude therefore that if the intelligibility of speech is a criterion of the importance of intensities in an audiogram scale that a stimulus scale is inadequate and that a sensation (or more correctly, log-stimulus) scale is satisfactory.

(b) It is evident that if the limit of accuracy of determination is measurable in terms of a certain definite stimulus change, regardless of the magnitude of stimulus, that the use of a logarithmic scale would be unjustified since points plotted at a value of the minimum accurately determinable value or less would be meaningless. According to Weber's Psychological

law, which holds particularly well in audition, the least perceptible change in a stimulus bears a constant ratio to the stimulus itself. This ratio may be taken as 5% in pressure change (See Knudsen in Phys. Rev., Mar., 1922). Assuming that the apparatus for varying the pressure is perfect, the accuracy of setting is directly related to the minimum perceptible change. Experience has indicated that without special effort, a setting is not dependable to within 20-30% on a pressure basis. This seems large, and it is large when regarded from the standpoint of pressure or stimulus on the drum membrane but exceedingly small from the standpoint of the sensation experienced. On the horizontal scale in Fig. 1 it corresponds to a width of about one tenth of a division.

From Fig. 1 it may be seen that an increase in stimulus pressure of from 1. to 1000, corresponds to a change in loudness of from something inaudible to a fairly loud sound, so loud in fact that speech at this intensity is entirely intelligible. If the same absolute stimulus change were made from 100,000 to 101,000, it would be imperceptible to the ear in accordance with Weber's law. It will be seen that the interval on the plot which corresponds to the first step (1: 1000) is large while that corresponding to the second step mentioned (100,000:101,000) is too small to be read.

From this it will be apparent that an audiogram plot on a stimulus basis (pressure or energy) is very misleading because of its magnification of the inaccuracies, and inadequate because it can only cover a small part of the sensation range. On the other hand, the logarithmic or sensation scale not only meets the requirements of uniformity of accuracy very closely throughout its range, but is the only simple scale capable of depicting all measurable characteristics. It follows that any scale of intensity which departs appreciably from this criterion has its utility needlessly impaired.

(c) From a purely dynamical standpoint the question of the most useful scale cannot be answered until the dynamics of the mechanism of the ear has been determined. In the somewhat similar problems of electro-dynamics and acoustics as encountered in telephone engineering the logarithmic scale has been found to answer the purpose much better than any other single scale. For the special case of the ear, of course, a special scale might be found to offer advantages of increased directness of correlation with certain lesions or variations in normal characteristics.

6. The Unit of Sensation.

From the standpoint of audiometry, the logarithmic scale is inevitable. Recent work by D. MacKenzie reported at the National Academy of Sciences, April 25, 1922 (The Relative Sensitivity of the Ear at Different Levels of Loudness) gives further demonstration of the utility of the use of the logarithmic scale as a scale of sensation or loudness. In Figure 1, the scale of intensity is the logarithm to the base 10 of the pressure.

In this paper the common logarithmic unit will be used as the unit of sensation. It corresponds to a change of pressure in the ratio 1:10 or energy 1:100. It will be seen from Figure 1, that the total range of sensation of the human ear for speech is perhaps a little over seven of these units. To make this more clear—suppose it were possible to attach a meter or indicating instrument to the auditory center of the brain which indicated on its dial the "loudness" of a sound stimulating the ear. If the stimulus of the speech wave were .001 dyne, the loudness meter would give no indication because it is inaudible. If the sound stimulus were 1. dyne, it would indicate just a bit less than three sensation units. If the sound stimulus were 1000 dynes, the sense meter would indicate just a little less than six units of sensation. Ordinary speech at a distance of three feet corresponds to a sensation of about three and one half units for the normal ear.

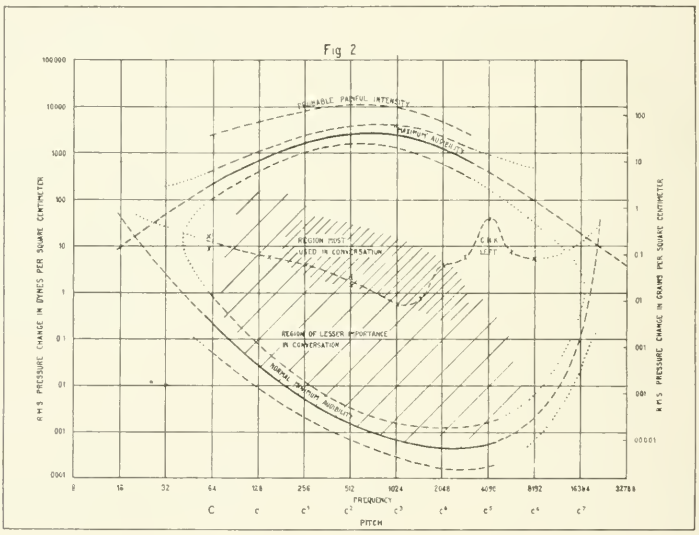
7. Minimum Audibility for Pure Tones.

Figure 2 shows a plot of the minimum audible pressure on the average of 72 normal ears taken throughout a range of frequency of from 60 to 4,000 cycles. Both the intensity and frequency scales are logarithmic. This curve has already been published (The Frequency Sensitivity of Normal Ears, by H. Fletcher and R. L. Wegel, Proceedings of the National Academy of Sciences, January, 1922, and in the Physical Review). The curve as originally published, was intended to represent the average telephone user. As a result of otological examination, certain individuals have been eliminated, and the average was found to be slightly different.

No normal ear has an audiogram represented by this smooth curve. Each individual audiogram has undulations which deviate from this average as much as ¼ unit. Variations in normal audiograms are considerable but the curves of hearing of the two ears of one subject are usually strikingly similar in many respects.

The different ways in which a definite lesion may affect a normal ear are probably at least as numerous as the anatomical and other variations in normal ears.

The statistical deviation from the mean varies irregularly with frequency very likely due mostly to the external anatomical variations which cause deviations in the dynamical constants of the transmission system from sound source to the ear drum. The broken lines following the curve of minimum audibility represent approximately the "standard deviation". A further discussion of the dynamics of these measurements was given in the paper referred to.



8. *Maximum Audibility for Pure Tones.*

The curve marked "Maximum audibility" represents the minimum average pressure stimulus on 48 normal ears required to produce the sensation of feeling. A sound much louder than this is painful. This curve was presented to the National Academy of Sciences in a paper "The Physical Examination of Hearing and Binaural Aids for the Deaf" by R. L. Wegel, April 25, 1922. The measurements were taken through a range of from 60 to 3,000 cycles. The standard deviation lines are also given from which it will be seen that this curve is quite as definite as that of minimum audibility. While this sensation of feeling is probably due to a stimulation of sensory nerves,

other than the auditory, it does serve as a practical limit to the range of auditory sensation. The few observations made indicate that people with abnormal ears have a point of feeling of the same order of magnitude as that of normal ears. Departures from normal "maximum audibility" or threshold of feeling sound vibrations are probably correlated with lesions of the middle ear which may not necessarily affect the auditory sense in any appreciable degree. The intensity necessary for feeling has been found to be about equal to that required to excite the tactile nerves in the finger tips.

9. *Lower and Upper Frequency Limits of Hearing.*

The curves of minimum and maximum audibility in Figure 2 will be seen to have been extrapolated to the points of intersection at high and low frequencies. The feeling sensation in the middle range of frequency at the threshold is a prickling sensation and then becomes acutely painful as the loudness is still further increased. The curve representing pain is, of course, rather indefinite and no attempt was made to measure it. Its probable position is indicated on the chart. As the frequency is decreased, the sensations of feeling become milder until at frequencies around 60 cycles, they are sensible as a flutter, normally quite distinguishable from the sense of audition. As the frequency is further decreased to a point where the hearing and feeling lines intersect, it is difficult to distinguish between the sense of hearing and that of feeling. The low point of intersection of two normal curves of minimum audibility and feeling sense may therefore be defined arbitrarily as the lower tone limit of audibility. The data indicate this point to be in the neighborhood of 20 cycles per second. For frequencies lower than this, the sense of feeling is more acute than that of audition. For any one individual this point cannot be picked with accuracy on account of the variation in judgment, but can be determined by extrapolation. A similar intersection of the two curves occurs at a very high frequency. The extrapolation upward of the curve of minimum audibility is in agreement with the observations of Mr. C. E. Lane of the University of Iowa. This work will be published shortly in the Physical Review. The upper tone limit seems to be in the neighborhood of 20,000 cycles.

We obtain by this reasoning, a rational way of defining the frequency limits of audibility and a method of making an accurate determination of them. Measurements of these limits which have been made in the past are questionable because the

effect of the intensity of the tone has been overlooked. Present generally available means for determining these limits are inadequate.

Near the lower limit of audibility the excursions of the middle ear structures are probably so large that the nerves in these parts are stimulated and therefore the measurements at very low frequencies probably give data related to the pathological condition of the apparatus of the middle ear independently of the stimulation of the auditory nerve. It is very likely that an "audiogram" of the threshold of feeling, plotted at all frequencies will be of significance pathologically.

10. *Area of Sensation.*

It will be seen that the area enclosed between the curves (figure 2) of minimum and maximum audibility as extrapolated defines the extent and absolute limits of the normal human auditory sense in both frequency and intensity. Any single frequency stimulus falling within this region corresponds to an auditory sensation. This portion of the area representing stimulus may be called the normal area of sensation.

Speech may be regarded as consisting of a large number of frequencies (band spectrum) stimulating the ear simultaneously and may be represented by a curve on the chart. If this curve falls within the sensation area, the sound is audible. The heavily shaded area in figure 2 is that most used in hearing ordinary conversation. (See I. B. Crandall and D. MacKenzie on "Analysis of the Energy Distribution in Speech", Physical Review, March, 1922).

For the sake of illustration an abnormal minimum audibility curve is drawn in figure 2. It will be seen that the sensation area in this case is much less than normal and that some of the speech region lies outside. This man understands speech with some difficulty.

11. *Accuracy of the Determination of the Normal Ear Curve.*

It is commonly thought that in order to obtain a normal ear curve, it is only necessary that a competent otologist pass on the condition of the ear before making the measurement and then to measure a large number of ears. A little examination into the errors entering into the determination of such a curve will show that this method is inadequate for an accurate specification of a "normal" ear. The measurement of any ear may differ from a hypothetical normal for a large number of reasons. Many of these tend to make the normal ear appear

low, a few to make it appear high, and others tend only to increase the variation among ears but these when taken in quantity do not affect the average. The problem is one principally for the statistician, but even though a normal curve in which all errors have been analyzed and eliminated has never been obtained, it is easy to obtain a serviceable normal with a comparatively small number of measurements. A table including most of the errors is given below. For convenience of analysis, these have been divided into six principal classes.

Errors in the Determination of a Normal Ear Curve.

1. Observational.
   - 1.1 Variation in position of sound source with respect to the ear.
   - 1.2 Variation in strength of sound source due to variable reaction of the ear.
   - 1.3 Error in reading instruments.
   - 1.4 Attention.
   - 1.5 Accommodation.
   - 1.6 Mentality of the observer.
   - 1.7 Variation due to technique of the operator.
   - 1.8 Method of setting apparatus for minimum audibility.
   - 1.9 Interfering noise.
2. Sampling.
   - 2.1 Variation with individuals.
   - 2.2 Variation with time.
   - 2.3 Experience.
3. Scale of intensity.
4. Variation with frequency and purity of tone.
5. Abnormality—uncertainty of detection of defects.
6. Age.

Of the errors mentioned above, some are larger than others and the relative proportions in which they enter into the determination of a curve depends on the circumstances under which the measurements are taken. In making the measurements recorded above, care was taken to minimize the influence of all of these errors and variations without actually making a quantitative investigation of each.

From the standpoint of the effect on the result, all errors may be classed as normal or skew, depending on whether they tend to cause greater variations about the mean without appreciably affecting its value, or to shift the value of the mean perceptibly.

If the skew errors could be reduced so as to be negligible, the problem of determination of the normal would be very simple since it could be determined with any desirable accuracy by making enough measurements. When, however, this cannot be done as in the case of the ear, the inference of accuracy from the average of a quantity of data however large is impossible. When skew errors cannot be physically eliminated they must be measured indirectly under properly controlled conditions and theoretically eliminated. In some cases it is difficult to determine whether an error should be classed as normal or skew without elaborate measurements.

Among the errors above, which are likely to be perceptibly skew the 1.4, 1.5, 1.6, 1.7, 1.9, 2.3 and 5. It is apparent that the problem of the accurate determination of the "normal ear" is difficult from the statistical standpoint.

As an indication of the order of magnitude of one of the skew errors (No. 5), the difference between random choice and otological choice of ears was examined, as well as could be done with the data without further tests. The "random choice" of subjects for test was made by simply choosing subjects who were not obviously "hard of hearing". The subjects were not even asked if they experienced difficulty in hearing. Otological examinations were then made of all whose audiograms fell below the mean of the first choice or showed unusual variations. Most of the others were also examined and found to be normal. Eliminations were then made of all showing abnormalities. While this is not a perfect "otological choice", it is very close to it from the standpoint of the result. A "frequency of occurrence" plot of the resulting readings of sensitivity was made (including all pitches in one curve) and it was found that the skewness, if any, was imperceptible and that the "standard deviation" was .33 units ($B1=.0059$, $B2=3.15$, No. of observations $=710$. A similar plot of the "random choice" data showed a decrease in the average sensitivity of .07 units, a standard deviation of .43 units and a quite perceptible skewness. While this cannot be said to be, in any sense, a determination of the error of abnormality present in a "random choice" it does show to what extent it is necessary to go to get a serviceable curve in terms of which to measure abnormal ears.

A "normal" ear has been observed to vary from day to day (see error 2.2) as much as one unit at 1200 cycles. Some ears vary more and others less. The variations are different at

different frequencies. Abnormal ears have been found in which the time variations are more of the order of a tenth of a unit (suggesting a degeneration of some accommodating apparatus). This indicates that the uncertainty of a single measurement of an ear is considerably greater in general than the difference, .07 units between the above named two choices, and that for a more careful determination of normal to be of any use a better technique of measurement and interpretation must be developed.

Other errors have not been investigated in as much detail. Errors 1.2 and 4. will be discussed under the heading "Audiometer". It might be noted in passing that the fact that the "frequency of occurrence" plot follows the normal error law is an indication (if proper reservations for the effects of other errors are kept in mind) that the error 3. "Scale of intensity" is eliminated by the use of the logarithmic scale. Any other scale would not give a normal symmetrical distribution about the mean. The ordinary uniform stimulus scale would be very much skewed. The study of the "normal" ear is a very large problem in itself and abnormal audition may be studied profitably without its complete solution.

12. *Useful Audiogram Scales.*

If it be accepted that logarithmic scales of both frequency and sensitivity (sensation) are the proper scales for audiograms, it must be decided in which form they are most useful. This depends of course on the use to which they are to be put. The form used in figure 2 is very good for demonstration of the physical aspects but probably inconvenient for recording routine measurements.

We have used two forms. The first is a plot at each frequency of the number of sensation units from normal audibility. This has the advantage of being simply related to the stimulus in that the pressure ratio to normal can be read directly.

The second form is that used in all audiograms given in this paper. From figure 2 it will be seen that the normal range of hearing at 1000 cycles (C3 is about 6.7 units of sensation). The illustrative audiogram in the same figure (CHK-left) has a range at the same frequency of about 3.9 units of sensation. This means (on the assumption of the logarithmic relation between stimulus and sensation and the acceptance of the end points) that CHK has approximately 60% (3.9/6.7) of the normal capacity to sense auditory stimulus. This capacity relative to normal is plotted in percent at various frequencies in Figure 3.

This chart gives perhaps the most practical and logical answer to the question so often asked by the patient "How much hearing have I left?" This can be read for single frequencies from the chart. The physician as well as the patient is usually interested in the loss, or amount retained of sensing capacity, which is of use in the interpretation of speech. It has been found (H. F. loc. cit.) that the most important range of frequencies for this use is from about 500 to 3000 cycles. A straight horizontal line through the mean of the audiogram included in this range gives the answer as well as can be done from an audiogram. Such a line has been drawn (dashed) in

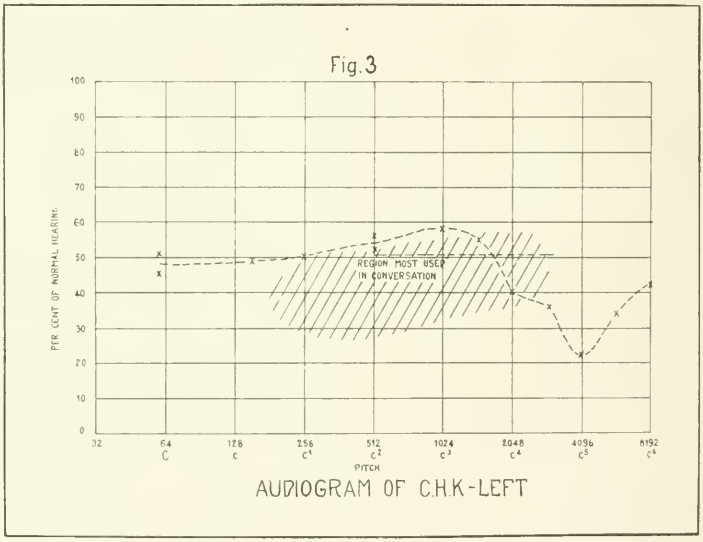


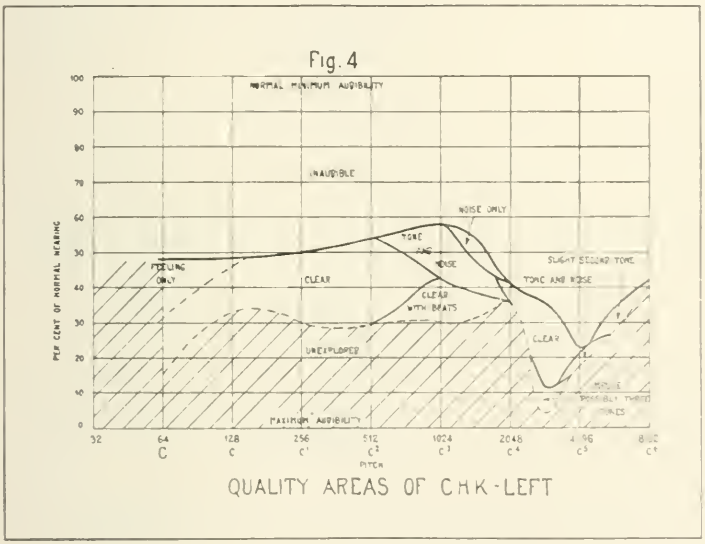
figure 3 from which it may be seen that CHK-left retains about 50% of his sensing capacity for speech.

13. *Quality of Hearing-Quality Areas.*

The quality of sensation of a normal ear at any point in the auditory sense area (Figure 2) may be described by any number of different adjectives, as for example, "clear", musical", "even", "sustained", "smooth", "pure", etc. Such a description may in fact be taken as a reasonable indication that the quality of sensation at the point in question is normal. Abnormal ears sometimes experience a subjective degeneration of quality of pure stimuli which they describe as "rough", "harsh", "sharp", "buzzing", "vibrating", "hissing", etc. These sounds are not

tinnitus. Figure 4 shows various regions of the sensation area which are degenerated in the case of CHK-left. The shaded area was not explored. The boundaries of the degenerated regions are usually more sharply marked than those of minimum audibility and the sensations in these areas are so radically different from the sensation of a pure tone that it is with difficulty that the patient is convinced that the stimulation is the same pure tone to which he has been listening at the other intensities. The subject of these tests is a violinist and capable of better descriptions and finer distinctions than the average.

Since all speech sounds may be considered as being composed



of various frequency components of certain intensities the sensation caused by such a sound may be represented on this plot by a line running through this area because the actual spectrum of the speech is a band spectrum. It is easy to see that if the points which represent those frequency components most essential to interpretation of the sound, fall within any of these abnormal areas, the sound is likely to be misinterpreted. Such people cannot understand many words which, nevertheless, they hear very well.

Many practically normal ears have very small abnormal areas. They have always been found near minimum audibility and therefore have little influence on the hearing. They seem to

be associated with so called catarrhal conditions although this cannot be stated positively. It is felt that they may prove to be of considerable pathological and prognostic significance. They cannot be detected satisfactorily except with very pure tones.

In order to assist the patient to describe the sound the following table has been prepared. This may also be used in determining the character of tinnitus.

1. Pure Tone	2. Vibration	3. Rattle
Smooth	Flutter	Gurgle
Clear	Trill	Rough
Musical	Shake	Clatter
Even	Wobble	Chatter
Bell	Tremolo	Discord
Whistle	Variation	Crackle
Sing	Irregular	
	Rythm	
4. Buzz	5. Hum	6. Roar
Bee	Rumble	Hoarse
Rough	Fan	Hollow
Sounds "Z", "ZH", "V"	Deep	Sea Waves
	Dull	
	Resonant	
	Sounds "M", "N", "NG"	
7. Sharp	8. Hiss	9. Harsh
Shrill	Swish	Rasp
Squeak	Rushing Steam	Sizzle
Edge on tone	Sounds "S", "SH", "F"	Frying
Piercing		Scratch

In addition to this the patient may be aided by being requested to compare the same pure tone when heard alternately in the two ears. If the ears are slightly different in sensitiveness only a slight difference in pitch will usually be described. A limited experience with these data indicates that two ears of a pair are usually very nearly alike as regards degenerated quality areas.

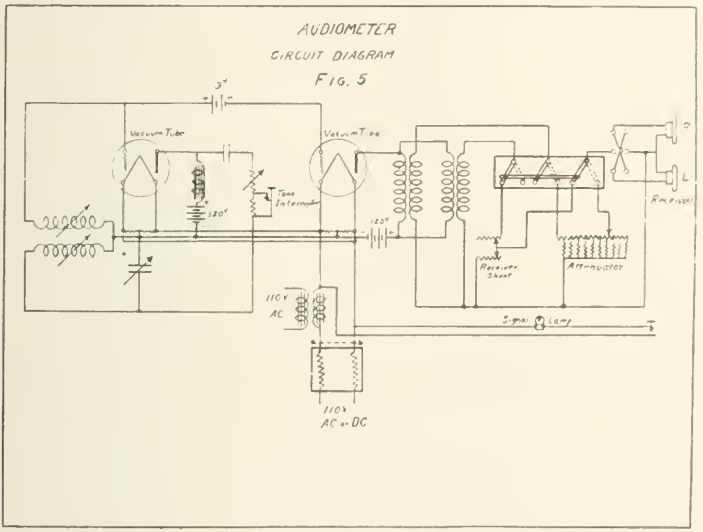
14. *Bone Conduction.*

If we may judge from experience with other complicated vibratory structures in acoustics and electro-dynamics, one kind of ear sensitivity curve, such as an audiogram, may not be sufficient for a complete dynamic analysis of the ear. The use of a supplementary audiogram made by means of a "bone conduction" telephone receiver will no doubt aid in diagnosis.

15. *Audiometer.*

Figure 5 is a schematic diagram of the audiometer used in the tests recorded here. It consists of a vacuum tube generator, a direct reading attenuator of the current generated, and a receiver designed particularly from the standpoint of constancy and for furnishing pure tones.

The oscillator circuit is capable of furnishing a very pure tone without wave filters, since particular care was taken in the selection of the circuit, coils and condensers. This is very important because throughout the lower portion of the frequency range, in particular, the harmonics of an impure tone are usually



more audible than the fundamental tone itself, so that the reading of minimum audibility depends on the sensitivity of the ear to the over tones and not to the fundamental. The criterion for purity of tone used was that the determination of minimum audibility should not be appreciably different for the tone as furnished by the oscillator than when subjected to a further purification by an electric wave filter. In order to meet this criterion, it was necessary that separate oscillating coils and condensers be used which necessitated the variation of frequency in steps rather than continuously.

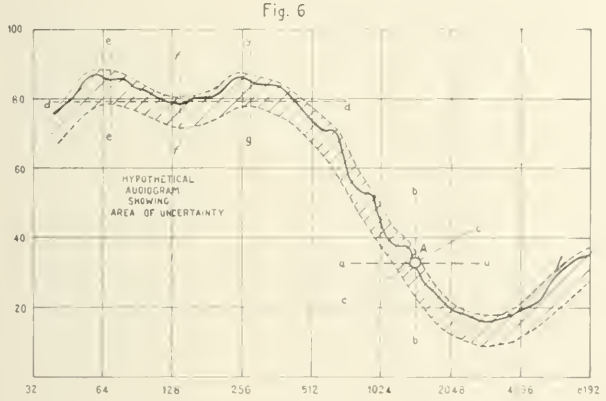
The taking of points at small frequency intervals, properly chosen, by varying the intensity and finding the minimum

audible point has an advantage over the method of varying the frequency. This can be seen by reference to the hypothetical audiogram shown in figure 6. A shaded area which includes the curve is shown. This area may be called the area of uncertainty. All points below the area representing loud sounds are points at which the subject very definitely hears the tone and all points above the area correspond to the intensities at which the subject quite certainly does not hear. Points in the area represent intensities at which there is more or less a degree of uncertainty as to whether the tone is heard or not. The only method of making an accurate determination of the audiogram shown is by taking a large number of readings at one frequency by varying the intensity, or a large number of readings at one intensity by varying the frequency and obtaining an average with sufficient precision for the purpose. Such careful curves have been made and the small irregularities found to exist. (See paper by H. F. and R. L. W. in Physical Review loc. cit.). It is important to point out that small irregularities are only determined by painstaking methods and that one measurement of a point on the curve is not sufficiently accurate. In general the width of the uncertain area is larger than the small irregularities indicated in the curve above referred to. We may, therefore, conclude that the only peaks or hollows in the curve which it is possible to obtain by single measurements, as is done in all present methods, are those which are as large as or greater than the width of the area of uncertainty. This width varies greatly with individuals and while it has not been carefully determined, it is estimated at about .2 units of sensation. This, in addition to the fact that no ear has been found which has a high sharp peak, leads us to conclude that readings taken at frequency intervals of about a half octave give a resulting curve with sufficient accuracy for present purposes.

The question of variation of intensity or frequency in making an audiogram can be answered by finding which, if either, most accurately, determines the curve. The accuracy of determination of any one point on the curve, for example the point A, figure 6, depends on how the sensation is varied in going through the area of uncertainty. For example, if the frequency is varied without the intensity, as represented by the line, aa, the error in the determination of the point is represented by the length of this line, aa, within the area. If the frequency is held

constant and the intensity varied, the error of determination of the point is proportional to the length of the line bb, included in the area of uncertainty. The most accurate method of determination of this point would be to vary both the intensity and frequency along the line cc, in which case the area of uncertainty is crossed by the shortest possible path. By similar reasoning, it can be seen that the determination of the top part of this curve is more accurately done by making determinations, ee, ff, gg than by varying the frequency at constant intensity along the line dd. In general, it may be said that these parts of the curve which have a slope within 45° of the horizontal are more

Fig. 6

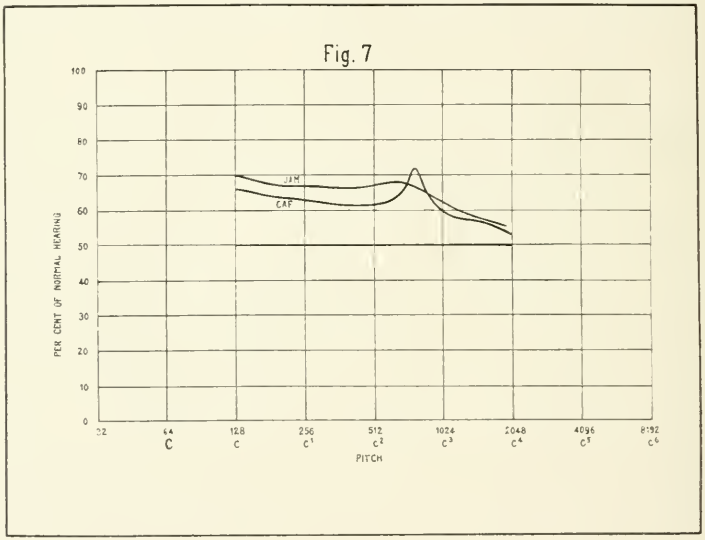


accurately determined by simply varying the intensities and those having a slope within 45 of the vertical are more accurately determined by varying the frequency. As a rule, the horizontal portions of the curve are of more importance than the vertical portions which may be considered more or less as transitions from one sensitivity at one set of frequencies to another sensitivity at a different range of frequencies.

The attenuator used is direct reading in logarithmic units and if the logarithmic relation between stimulus and sensation be assumed, it may be considered to read directly in units of sensation rather than stimulus. This attenuator is a low-resistance iterated resistance network developed from the point

of view of avoiding interference between its input and output ends. The low resistance admits of a more effective elimination of this interference by the suppression of undesirable capacity effects. The total attenuation of the apparatus used was such as to vary the sound intensity (pressure or receiver current) at a ratio of about 1:10,000.00. It is impossible to obtain this amount of attenuation without special regard to the interfering effect of stray capacities.

The receiver used is one in which the diaphragm is damped by air damping (I. B. Crandall, Phys. Rev. June 1918) to such a degree that the effect on the diaphragm, of placing it on the



ear is negligible. Figure 7 illustrates the effect of different ears on an ordinary telephone receiver diaphragm. The two curves plotted on a "percent hearing" scale give the comparison of an ordinary commercial telephone receiver with a highly damped receiver by two individuals. It is easy to see that regardless of how accurately the average minimum audible point is determined with such a receiver, each audiogram must show a peak or at least an increase in sensitivity which is due to the diaphragm only and therefore must tend to uncertainty in interpretation. In addition, the natural frequency of telephone receivers is usually in the region of from 800 to 1200 cycles per second and even with an oscillator having a comparatively pure

frequency, the small residual harmonics which always exist are greatly magnified if they fall within the region of resonance of the diaphragm. This means that determinations of minimum audible points below resonance of the diaphragm are likely to depend very largely on the sensitivity to harmonics rather than to the fundamental frequency. These errors, 1.2 and 4 cannot be eliminated by averaging.

In this connection it may be noted that a similar error (1.) due to the acoustic effect of anatomical variations in external ears can only be eliminated by an acoustic study of the subject. The variations due to this cause are comparatively small at intermediate or speech frequencies but increase at very low and at very high frequencies. This cause is no doubt responsible for many of the smaller peculiarities at high frequencies. The practical conclusion to be drawn is that only the very large peaks or hollows at these frequencies can be considered as having pathological significance. As an error in the normal curve, it cannot be known whether it is skew or not without acoustic study.

Conclusions of Part I.

1. In an audiogram the most serviceable scale of intensity is proportional to the logarithm of the stimulus. Any other scale has a needlessly impaired utility. The best scale of frequency or pitch is in all probability also logarithmic. The musical scale is the best known example.

2. The range of stimulus of pure tones which the ear perceives is limited on the lower side by the minimum audible intensity and on the upper side by the sense of feeling.

3. The lower and upper frequency limits of hearing may be defined as those pitches at which the sound stimuli corresponding to the thresholds of audition and of feeling are equal. For frequencies below the lower limit and above the upper limit the acuity of the feeling sense is greater than that of the auditory sense. For frequencies between these limits the auditory sense is more acute than that of feeling.

4. A serviceable "normal ear" curve can be obtained by a small number of measurements without extreme care in selection.

5. The ideal "normal ear" can only be determined by the coordination, by a statistician, of data obtained from the otologist, psychologist, and physicist.

6. A plotting of the "per cent" of hearing or of normal sensing capacity is logical.

7. The quality of sensation may prove to be of an importance comparable with the sensing capacity or acuity.

8. Bone conduction graphs should be of great value.

9. A graph of threshold of feeling may be of use especially in middle ear diagnosis.

10. The tone from an oscillator should be so pure that the error in reading due to the presence of harmonics is less than the observational error.

11. A current attenuator should be free from interference between the initial and attenuated currents.

12. Most commercial telephone receivers, when used in an audiometer, introduce intolerable errors.

Part II. Application

Unlesss we approach the study of the audiograms made by the methods we have detailed with a mind free from the bias of preconceived ideas and current teaching on the functional tests, we are destined to fall into grave errors of interpretation, and, by so much, fail in advancing the knowledge of audiometry. Let us, then, at this time, confine our efforts to a statement of facts—facts shown so clearly by the graphs that all may read and study them.

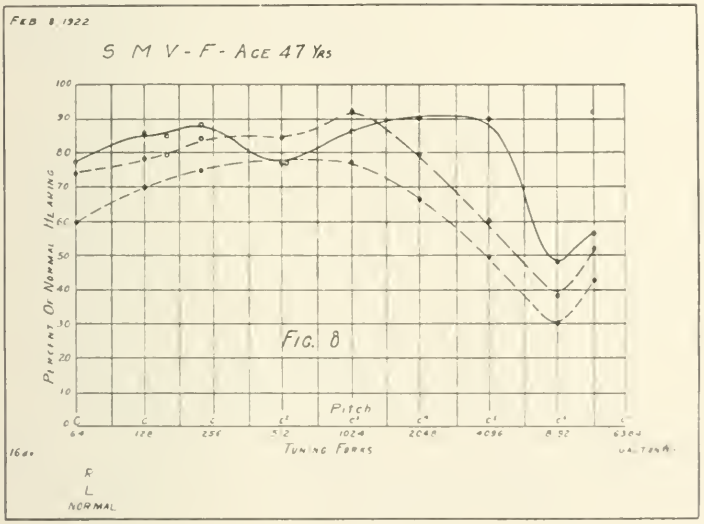
The usual tests by tuning forks and Galton Whistle are given beneath each chart, not so much for possible coordination therewith, as to show how more definite and reliable are the audiograms, because each point determining the curve denotes a definite frequency and intensity.

For clearness we have omitted frequency figures at the one half and one third octave points. In all instances where these were taken they confirm our opinions as to their needlessness for a better determination of the graph; the audiograms were chosen to demonstrate several types of curve and lesion. Charts showing the effects of experimental dulling of the hearing with cotton and vaseline plugs in the external meatus show the resulting changes in the transmission characteristics of the ear to be variable with frequency, but not according to any simple law. The average normal curve is represented in the graph by a straight line marked 100% hearing. The use of the straight line possesses many advantages.

Case Reports

Figure 8. Effect upon the hearing of occlusion of the external auditory meatus. In the left ear there is a deformity of the posterior meatal wall which when the jaws are closed or the pinna is pressed in and forward, completely occludes the canal by closely approximating its posterior to its anterior margin. The loss of hearing is annoying and in the graph appears to vary from 7% to 15%, depending upon the frequency. Neither ear is normal, but the hearing is fair as indicated by the 80% to 90% remaining in the most important area for speech (500 to 3,000 cycles).

Note (a) the similarity of form of the two curves (b) their crossing at 300 and 1,400 cycles, (c) the persistance of similarity throughout the frequency range while the meatus is occluded and the slight differences in percentage of loss of hearing at all frequencies.



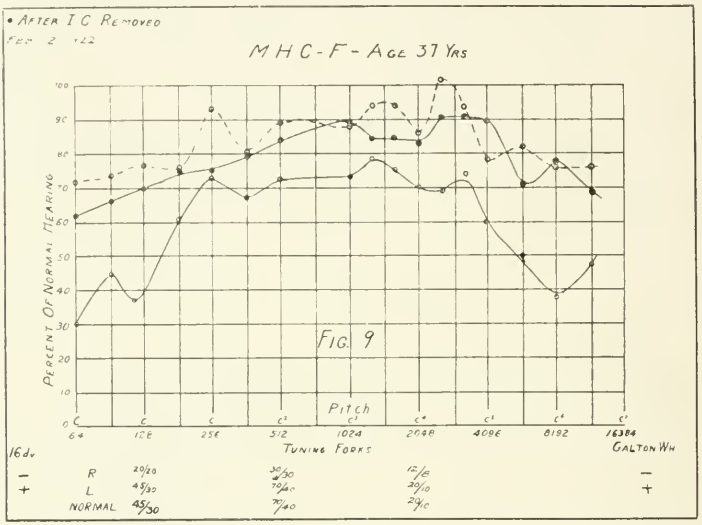
(d) The evenness of the curves. This general evenness with many small undulations appears to be a normal characteristic of the human ear.

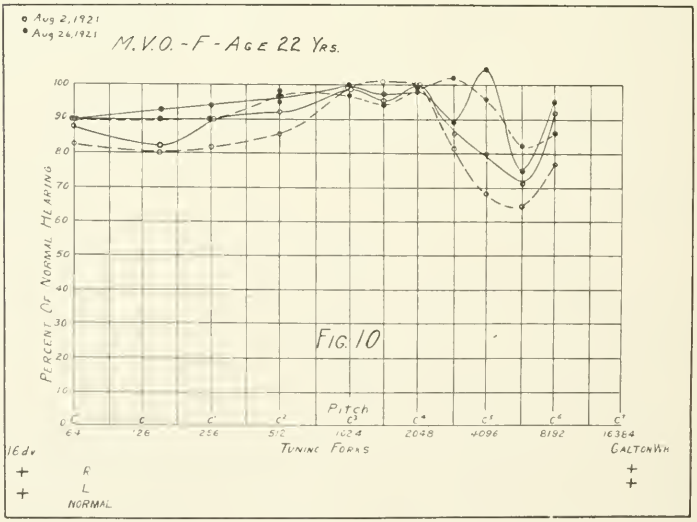
(e) The grade of the slopes in the low and high ranges—the brow of the slopes of the two ears relative to frequency.

Figure 9. Effect upon the hearing of the removal of impacted cerumen which completely blocked the canal. Improvement in hearing occurred throughout the frequency scale but greater in both the low and high registers than in mid range. A constant ringing tinnitus ceased thereafter. Both ears are abnormal but the hearing for speech is excellent as it should be on account of the high capacity in the speech area. All tones appeared muffled in the right ear before the I. C. was removed—thereafter clear. Tinnitus was similar to 262 cycles. The Galton and 16 DV fork were heard O. K. at second testing.

Note (a) similarity of curves as to form even before the wax was removed from the right ear. (b) Lack of marked peaks and hollows. Near coincidence of the two curves after the obstruction was removed.

(c) Relative height of the sensing capacity at mid range as compared





with the depressions at both ends of the scale.

(d) The marked lowering of the curve, especially at high and low frequencies by the obstruction and the subsequent elevation in both of these registers.

Figure 10. Improvement following treatment in case of O. M. C. C. R. and O. M. C. Ac. L.

The patient thought there was little change in the right ear but a change from very deaf to normal in the left ear.

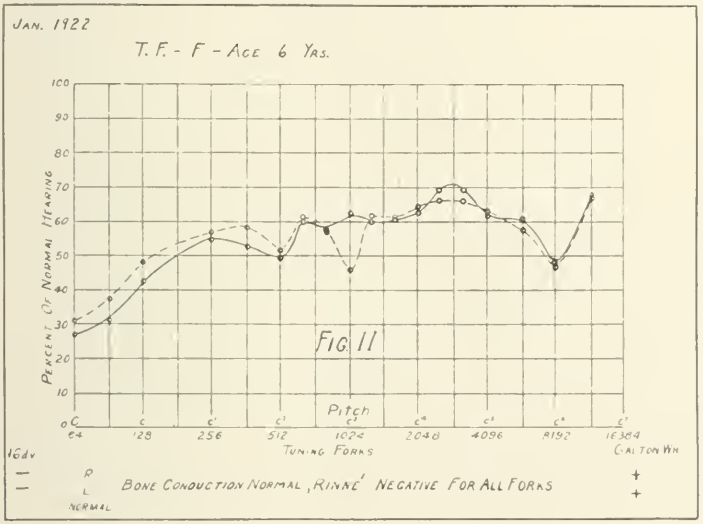
A constant tinnitus in the left ear at the time of first testing had permanently disappeared at second testing as did many subjective tones elicited at certain frequencies by the lower intensities.

Note (a) similarity of form of curves of the two ears and its persistence during the inflammation.

(b) The evenness and close coincidence of the curves of the two ears for the greater part of their length.

(c) The tendency of the high capacity at mid range to persist.

(d) At both the low and high registers the improvement following treatment for obstructive deafness. In this case does the lowered hearing of from



20 to 25% at 6,000 cycles indicate a lesion in the nerve or end organ? This cannot be stated, nor in many cases showing similar variations around this frequency as may be observed. A purely obstructive lesion may cause a like fall in hearing. The occurrence of subjective tones elicited by the lower intensities may possibly be explainable on a purely mechanical basis as due to conductive mechanism vibratory abnormalities.

Figure 11. O. M. S. C. recurrent—both ears, of five years standing, at present healed.

The middle ear spaces are largely obliterated by greatly retracted drums and masses of scar tissue with areas of tissue thinness of low tension and limited mobility. Mallei immobile, tubes fairly patent. No vacuae observed in middle ears except before acute exacerbations or recurrences. Hearing for whisper varies from 10 to 20 feet in both ears depending upon foregoing conditions. It appears to be a case of marked obstructive deafness.

Note (a) similarity, evenness and coincidence of curves, (b) tendency of percentage of mid register to persist. (c) Very marked and progressive depression toward lower frequencies.

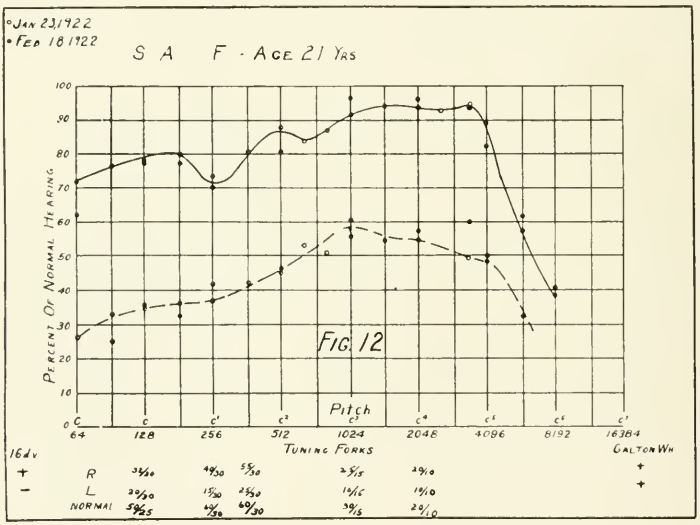
(d) No tendency to approach normal at any frequency. Does the loss of hearing in the upper register denote nerve deafness? This cannot be positively stated as purely obstructive lesions may cause a similar loss in these frequencies.

Figure 12. O. M. S. C. healed for one year, right.

Radical mastoid healed two months, left.

Hearing for speech good in the right ear. For whisper 30 feet. In left ear whisper heard one foot.

The data taken on February 18th is shown without a curve being drawn. It differed but slightly from the prior plotting although the patient stated she



was hearing better at the later date.

Note (a) similarity as to form of the curves in the two ears.

(b) The persistence of this similarity in these two ears in spite of the dissimilarity of the lesions (c) the evenness of the curves (d) the tendency of persistence of high capacity at the mid ranges (e) no approach to normal in the poorer ear (f) the precipitous drop in both curves near 4,000 cycles. The difference between the percentage of these drops between 4,000 and 6,000 cycles.

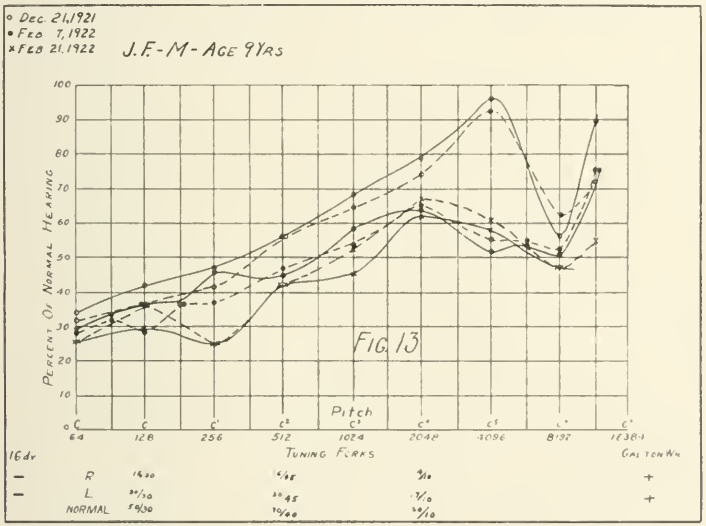
Does such a percipitous and extensive drop indicate nerve or end organ deafness? From present knowledge it cannot be stated that it necessarily does so.

Figure 13. Radical mastoid right, healed.

O. M. S. C. left profuse discharge with small remnant of drum remaining. Middle ear structures greatly congested. No labyrinth symptoms.

The hearing in both ears is diminishing as shown by the first and second tests and also in another test omitted from the graph to avoid multiplicity of tracings. The February 21st graphs were made while the right ear was plugged with simple cerate and the left contained considerable pus. This caused a loss of hearing in the plugged ear, especially at 250 cycles and 1,000 cycles. The high per cent of hearing at 4,000 cycles on December 21st may be due to the fact that the operated ear has just healed and the running ear had been asperated and cleansed.

Note (a) the general slope of the curve from 60 cycles up to 4,000 cycles at first testing. (b) The similarity and near coincidence and persistence of these similarities in all the tests and in two ears differing greatly in their anatomical and diseased conditions. (c) The evenness and tendency of the curve to remain high at the middle frequency.



(d) The rapid drop from 4,000 cycles at the first reading and the smaller changes at 60 and 8,000 cycles—also the relatively high capacity at 11,000 cycles.

(e) Is there anything in these curves to warrant the diagnosis of a complicating nerve lesion?

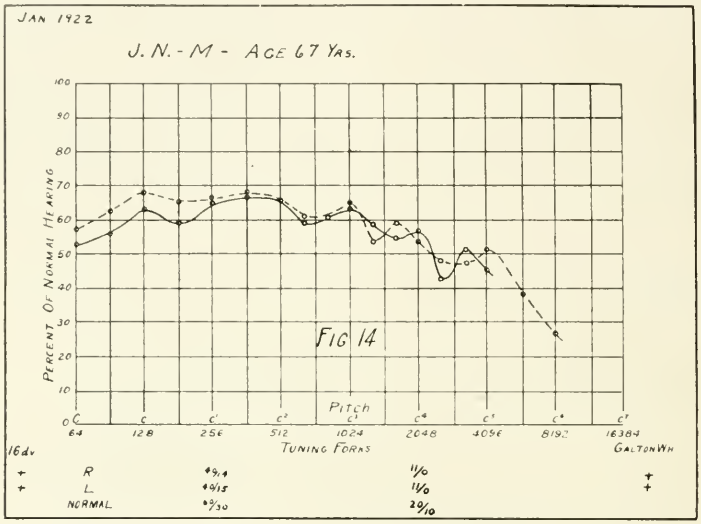
(f) The definite charting of the continued progress of this case should give valuable data and in this connection the change in position of the high point between the first and subsequent testings should be noted.

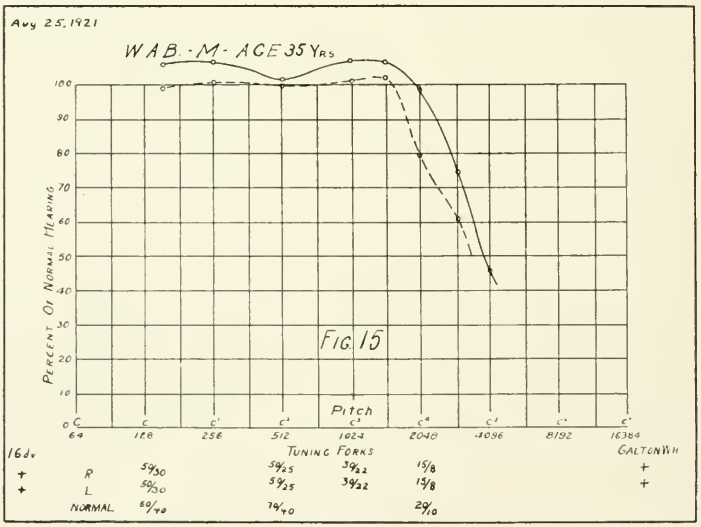
Figure 14. Senile progressive deafness—10 years duration.

No history of other symptoms of ear disease. Otoscopic findings negative. Tubes patent. No vacuae in middle ears. Inflations did not change the acuity of hearing. He claims to hear Galton O. K. but it may be the true tone was not sensed. The 16 DV fork was heard and felt. It is doubtful which sensation predominated.

Note (a) similarity, evenness and near coincidence of curves. (b) Lack of high sensitivity at any frequency. (c) No precipitous drops. (d) The general slope of the curve downward from low to high frequencies.

Figure 15. Progressive deafness in both ears following acute catarrhal





otitis in the left ear after swimming, nine years ago. Treatment temporarily relieved the blockage but he dates the onset at this time. Mother, sister and grandmother deaf for years. Intermittent ringing tinnitus right ear. No labyrinth symptoms. Both drums retracted, left, thin; right, dull and opaque.

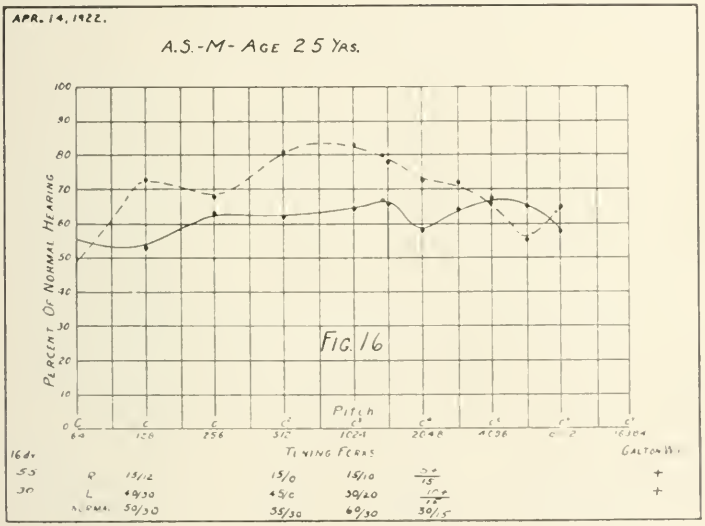
No vacuae in middle ears.

Watch heard R. 3″; L. 1″.

Whisper R. 15 feet; L. 6 feet.

Note—the loss of hearing in the left ear from 1,500 cycles and in the right ear from 2,000 cycles up. Both ears lack good hearing in the upper part of the speech area. Speech of late is becoming more and more difficult to understand. Should subsequent examinations find the brow of the curves receding towards the lower frequencies with a gradual straightening out of the whole curve, it is probable that the rate and amount of such changes in form should be of value in diagnosis and prognosis. Instances of this phenomena have been noted in cases of progressive deafness which were difficult to diagnose from information derived from present tests.

Note (a) the similarity, evenness and high capacities at all frequencies



up to 1,500 and 2,000 cycles at which points precipitous drops occur. Had it been possible to produce tones of greater intensity than the two higher octaves, this patient might have heard them. Lack of charting these tones means only that the telephone receiver is not efficient enough at high frequencies and not that these frequencies cannot be heard. The measuring of acuity of hearing by frequencies, irrespective of their intensities is, of course, crude.

Figure 16. Syphilitic deafness—first noticed in the right ear ten days before the audiogram was made. No prior ear disease. All the classical signs of syphilis eight months ago. Wassermann ++++. Four subsequent negative tests, the last two weeks ago. Has had intensive treatment with neosalvarsan and Hg. Otoscopic examination negative. Tubes patent. No vacuae, inflations give no relief.

Whisper R. 1″; L. 15″.

Watch R. & L. + tragus—mastoid.

Weber to better ear.

No spontaneous nystagmus.

After rotation to R. nystagmus 16 seconds.

After rotation to L. nystagmus 9 seconds.

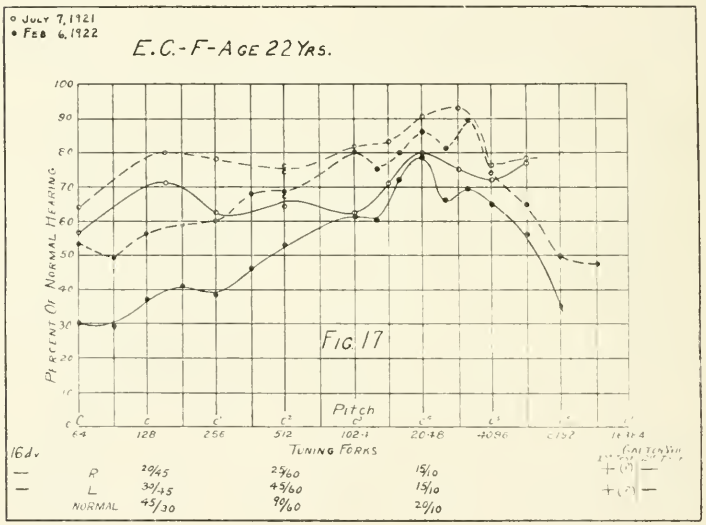
Vertigo coincides with nystagmus.

Caloric after 50 seconds lasting 1½ minutes.

Normal changes with head in all the different positions. Past pointing normal. Lower frequencies appear one tone higher in the left ear. Higher frequencies appear one octave higher in the left ear. Note (a) general similarity and particular dissimilarities in form of the two curves.

(b) Tendency for the high capacity to persist only in the middle registers in the left ear.

(c) No precipitous drops in either curve. (d) Percentage of loss of



hearing about the same at all frequencies for the right ear. As an aid to the diagnosis of syphilitic affections of the ear audiograms should be of value if taken at proper intervals to show the rate and peculiarities of the progress of the disease.

Figure 17. Progressive deafness noticed suddenly one year ago following a hysterical crying spell.

Tinnitus constant in right ear followed in a few weeks by a like tinnitus in the left. Right ear very deaf ever since and left somewhat affected. Vertex headaches at times. Otoscopy shows drums retracted thin and tension diminished. No vacuae in middle ears. No ankylosis or scars.

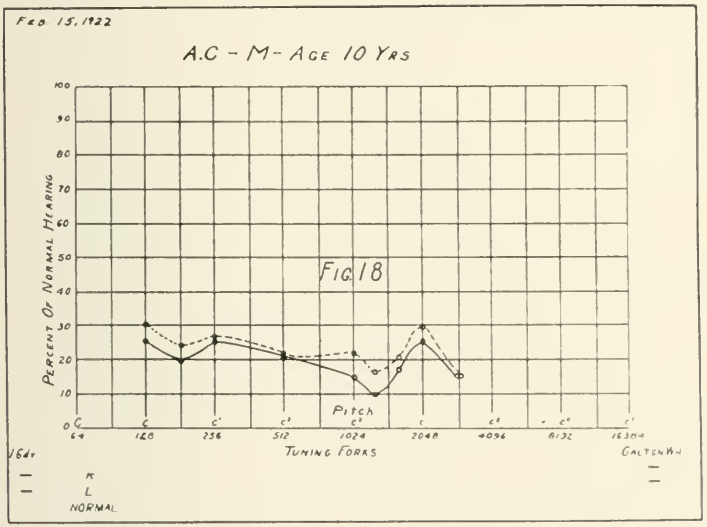
Shrapnell's membrane in right slightly congested. Thyroid gland somewhat enlarged. Wassermann negative. The interval between the two tests was seven months. It is questionable if the Galton was heard at the first testing. It was not heard at the second. Hearing for ordinary speech is getting worse

owing to the loss of capacity in the speech areas. Fowler's modification of Gelé*.

Note (a) similarity of curves as to general form. (b) Persistence of this similarity at the second testing although in the interim a marked drop had occurred in both upper and lower frequencies and the difference in capacity as between the two ears had increased especially in the lower half of the frequency range. The near coincidence of the curve in some of the middle frequencies and the tendency of the capacity to persist between 1,000 and 4,000 cycles.

(c) The almost percipitous drop at about 3,000 cycles in the left ear carried further down in the second test.

(d) The position of the brow of the curve in the right ear as compared with that of the better left ear.



(e) The loss in the higher frequencies accentuated at the second test. At certain distances from minimum audibility at all frequencies at both testings, tones sounded to the right ear rough or muffled—to the left ear, clear.

Further tests at intervals of a few months will be taken and the charts studied particularly to determine the rate and peculiarities of the progress of the case.

Figure 18. Congenital Deafmutism.

Prior to 1½ years ago heard nothing (?). Is slowly acquiring a vocabulary by mimicing very loud speech heard at a distance of one foot. Can understand without reading the lips the limited vocabulary learned.

Note (a) At these extremely low capacities we find in many cases a similarity of the curves of the two ears.

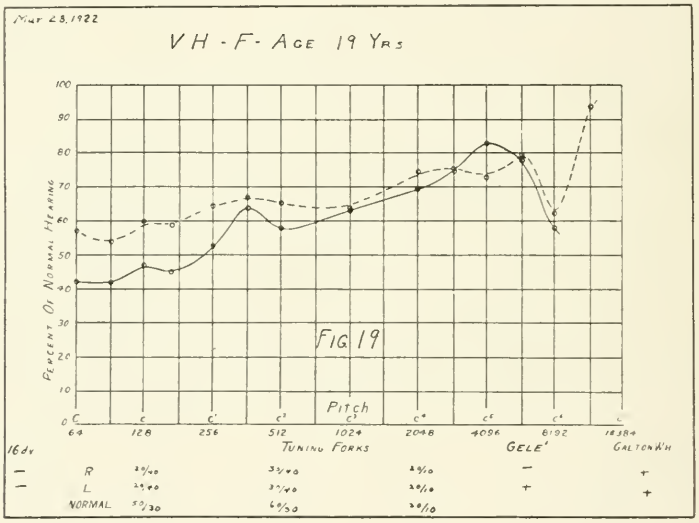
(b) Evenness and near coincidence of the curves. (c) The advantage of definitely determining even this residual capacity in deaf mutes is apparent.

(d) The words learned by the sound of speech, though they are but partially heard, sound like a different language compared to those acquired by lip reading.

By what sequence of events did these ears descend so low in the scale of percentage of hearing? When we have followed curves from near 100% down through the various consecutive changes in plotting to near the zero mark, valuable data will be obtained as to the sequence of events accompanying loss of hearing.

Figure 19. Otosclerosis.

Family history of progressive deafness—first noticed right ear was defective six years ago. Shortly thereafter the left ear followed suit and to date both have slowly but surely lost capacity for sensing sounds, the right always being the poorer ear. No prior history of ear trouble except possibly earache in



infancy. Otoscopy shows drums slightly retracted opaque and luster diminished. No congestion, freely movable. Light reflexes fair. Mallei somewhat immobile. No vacuae. Tonsils and adenoids removed and inflations used without arresting the deafness.

Voice R. 12″; L. 15″.

Whisper R. 7″; L. 10″.

Watch R. 3″; L. 8″.

Static labyrinth normal. Note the similarity in form, evenness and near coincidence of the curves in middle frequencies. The general slope of the curve upward and to the right where the left reaches near 100%.

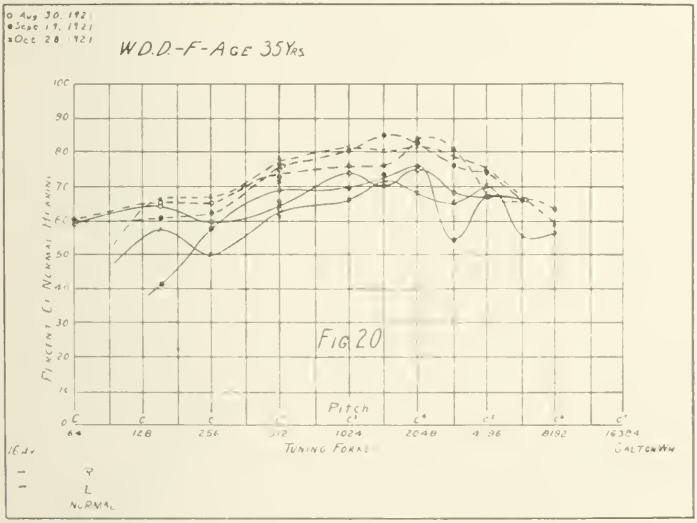
The suggestion of a beginning percipitous drop at 6,000 cycles. Fowler's modification of the Gelé was used. The standard method failed to elicit response in either ear. Audiograms at stated intervals after sudden changes in hearing will be of interest in this and similar cases.

Figure 20. Three audiograms taken at different times showing the percentage of hearing to be practically identical in each ear.

This patient claimed that at the second test her deafness was cured and at the last test her hearing was very poor. There did not appear to be any improvement in the hearing for speech and the audiograms confirmed this opinion much to the disappointment of the patient. At the first test the patient suffered from severe tinnitus of predominating frequency of 100-200 cycles. At the third test the pitch of the tinnitus had increased. Certain tones in one ear temporarily annulled the tinnitus. In the other ear they were without effect.

Note (a) the near coincidence of all the curves of the left ear and the slight variations between the three curves of the right.

(b) Similarity, evenness and tendency of persistence of the middle registers.



(c) Lack of precipitous drops. The value of audiograms as a means to check up notions as to losses or gains in hearing and especially if attributed to treatment.

Attention is called particularly at this time to the following striking characteristics of these curves, and a few inferences are drawn from them. Other peculiarities and deductions will no doubt also be suggested. These striking characteristics are not limited to the charts shown, but hold true with very few exceptions for all those made.

(1) Similarity as to general form of the curves of the two ears in each subject tested.

(2) The persistence of this similarity of form, irrespective

of differences in lesions and in per cent of hearing retained in the two ears (also in deaf mutes).

This strongly suggests that every pair of ears normally shows a certain pattern of graph, and the possibility of using this pattern as a base upon which to correlate our findings in respect to anatomical, physiological and pathological peculiarities in the ears examined.

(3) Evenness of the curve—no inordinate dips or rises (gaps and island). This appears to be characteristic of the human ear.

(4) The close coincidence not only of form but of percent of hearing of the two ears in certain cases in similar and also in quite dissimilar lesions.

(5) Tendency of the persistence of high relative sensing capacity in the middle register.

This hanging on to the area most useful in speech accounts for the remarkably good hearing often possessed by persons who have marked defects in their hearing for sounds of other frequencies. It would seem as though the speech areas were made most resistant to disease processes, and that we should look for incipient or slight defects, particularly in the upper and lower frequencies.

(6) When a graph shows a low sensing capacity through a considerable frequency range there is seldom normal or hyperacuity at any frequency. In other words, a marked lowering of the hearing is seldom accompanied by normal or high acuity for any frequency. The reverse does not hold true.

(7) Precipitous drops occur almost wholly in the upper register (from 3000 up). The position of this (brow of the hill) point of sudden descent as to frequency and intensity will be studied in relation to its diagnostic and prognostic significance, and also as to the rate and degree of the shifting of its position.

In certain cases there appears to be a gradual shifting at this point toward the lower frequency, coincident or not with its descent in the intensity scale.

(8) Loss of hearing in the higher notes occurs not only with nerve deafness but with certain so-called obstructive deafness. We have been so accustomed to think of loss of hearing for the higher tones as meaning nerve deafness that it is well to pause and realize that this is not necessarily true. It is easy to prove that any high tone, and even the highest normally audible tone, will not be heard unless it possesses sufficient intensity so that if the intensity of such a tone is

prevented, by an obstructive lesion, from reaching the ear in sufficient force (a pressure equal at least to minimum audibility pressure for such tone) the ear will not sense it. Experimentally produced and natural lesions are shown by some of our curves to markedly lower the sensing capacity of the ear for the highest frequencies in our scale, the Galton Whistle can be used to demonstrate the same truth by lowering its intensity by distance—some people with normal hearing cannot hear the whistle close to the ear if the latter is held closed by the finger.

(9) The possibility of accurately noting the rate and peculiarities of progressive loss or regaining of hearing—this data should be of great value to the otologist.

(10) The value of accurate audiograms to check up notions as to improvement or loss of hearing following treatment.

(11) The possibility of aid to the deaf mute who has a little hearing left in the speech range. Several children tested are now being taught by properly accentuated speech.

The possibility of using the graph as a basis for building deaf sets to bring defective speech areas up to a given level, so that speech may be more perfectly understood, instead of simply increasing equally the loudness of all the sounds transmitted to the ear.

DISCUSSION.

Dr. L. W. Dean, Iowa City, Iowa, said that he had compared Dr. Fowler's work with his own and they had gone over many more details than had been dealt with to-day. The society should be congratulated that Dr. Fowler was doing this type of work. Dr. Dean said he thought it could not be laid to over enthusiasm if he said that from the type of machine Dr. Fowler is working with, and the type envolved in their work at Iowa, they would get an instrument that would have practical and scientific benefit. At the present time it is more scientific than practical. The method of showing diagramatically the extent of the tonal range would be of more value later after study and interpretation. Of 150 charts of Dr. Fowler's, Dr. Dean said he had missed the significance of the curve in only one case. Probably, however, he had more practice in interpreting the curves, than one who was not familiar with them. There was no doubt, however, that the work done by Drs. Fowler and Wilson, and that done at Iowa would be recognized to have clinical and pathological significance.

Dr. Max Goldstein, St. Louis, Mo. said that while he was not unmindful of the scientific and research value of these charts, he was not ready to forsake older methods of testing for diagnostic purposes. These charts contributed to the diagnosis as does a temperature chart, but if one used the temperature chart to judge the necessity of mastoid operations, one would be handicapped. Of the two machines of Drs. Dean and Fowler, the speaker said he believed he would use the one originated at the University of Iowa. The objection to Dr. Fowler's was that he used the telephone receiver to recive the sound, and this really muffles the sound. With the forks one gets from 64 to 32 thousand double vibrations, with the whistle 64 thousand. With the Dean instrument one has from 40 to 75 thousand and from 20 to 18 thousand.

To test acuity for the upper tone, the monochord can be used. The audiometer is of less use than bone conduction. In the mastoid cases with the Weber fork on the vertex the sound is lateralized to the opposite. Later it comes to the good side. There is further invasion of the operated ear. That suggests looking for labyrinthine invasion. One does not get that information with the audiometer.

Mr. Fletcher of the Western Electric Co., New York City (by invitation) said that the impressions of an outsider might be useful to the Society. He wanted to ask some questions. First, Why speak about use of the voice? The voice is not used really; the frequencies are different. All the tests are based on the whisper. Some apparatus should be made in which the voice could be used, and the voice varied. Physicists should be able to make some such device, one in which the voice is made to go down till the patient heard it. That should be simple. Otologists had got used to certain tests and did not like to give them up. Another point: In regard to the forks; how hard should one hit the forks? Also there is a great deal of difference in the length of vibration of forks. Another point is the influence of the room: in the open air the fork sounds differently from the sound in the room. This is more apparent with the whisper than with the voice. There is fifty times the pressure in a noisy room as in a quiet place. The acoustic properties of the room make all the difference in the world.

In regard to the value of the audiometer, the Western Electric Company had been forced to meet otologists and physicians. The telephone was familiar to them, but it did not occur to them that speaking was the beginning and hearing was the end of it and that the voice was important. In studying the question of hearing, they ran into the problem of abnormal hearing, and the need of measurement was felt. The Company would no doubt be able to meet the needs of physicians, when the need becomes clear. The diagnostic value of audiometers is one side. There are some kinds of deafness one can do something for and one should know what they are. The Company should be able to construct an audiometer for fifty dollars that could be put in every school so that all the children could be put through the tests and the abnormal child will be brought to the otologist to see if anything can be done for him. Physicists should, by knowing the principles involved, be able to make deaf sets prescribed by the physicians, and also to make the audiometers for the physicians so that they can prescribe the deaf sets when necessary.

Dr. George E. Shambaugh, Chicago, Ill., said that the last speaker had touched upon the practical side of this method of testing. It would show what part of the tonal range needed assistance, and what the instrument makers could do to provide the necessary assistance. The audiometer does not help in the fundamental problem as to whether the patient will receive any help by treatment. The otologist can make this diagnosis very readily by other means, than by running through the tonal range. The voice tests are of greater value. In cases of nerve deafness the parts of the tonal range which are destroyed may be picked out by the audiometer, and in this will lie its chief field of use. The mechanical inventor can devise means of assisting the parts of the range which are destroyed.

Dr. E. P. Fowler (closing) said that he had neglected, for lack of time, to read the conclusions of the paper. In the conclusions he had taken up all these points. One point was, were otologists sure that the tests they used, meant what they supposed they meant? The more he had become interested in these accurate methods, the more he had come to doubt the surety of the others. One could not predict exactly what the charts mean but they are certainly very suggestive. In time and with further study the interpretation would be more clear. A method of applying bone conduction with the instrument, with definite frequencies and intensities would be worked out, and then two crosses could be made, one for air conduction and one for bone conduction.

CASE OF POLYARTHRITIS COMPLICATING MASTOIDITIS. OPERATION. RECOVERY.

By DUNBAR ROY, M. D., Atlanta, Ga.

Focal infections as a cause of arthritis seem now to be an established fact. However mastoiditis whether acute or chronic seems to have received but little mention as one of these causes if we may judge from the infrequency of reported cases.

While various conditions such as glycosuria, convulsions, and other nervous phenomena often occur in the course of acute infections of the ear and while there seems to be no good reason why rheumatism, arthritis and neuritis should not occur from acute and chronic ear affections, yet the complications which do occur are the conditions which are usually denominated an pyemic.

In looking over my own personal histories of aural diseases for the last twenty five years the writer has been unable to find any other case even resembling the one here reported and the literature examined would certainly lead one to the conclusion that such a complication is exceedingly rare. What few references as have been found have been reported in this country so that one is forced to the conclusion that such complications are so frequent as not to be worthy of mention or that has not been observed.

CASE HISTORY.

Jack J. White, age 4. Strong, healthy looking child giving only the history of repeated colds in the head.

Examination shows tonsils very much enlarged and also the presence of increased amount of adenoid tissue. Operation advised and on Feb. 2, 1921 at Georgia Baptist Hospital both tonsils and adenoids were thoroughly removed by my associate Dr. Murdock Equen. Convalescence was uneventful and in a few days the child was out and immediately began to improve.

On March 5th, 1921, nearly a month after the tonsil and adenoid operation, the patient began to complain of severe pain in right ear and two days later the left ear also began to pain.

Dr. Equen was called in consultation and finding both ear drums red and bulging, under chloroform anesthesia, these were freely incised. This was followed by a free discharge of mucopus from both middle ears, all pain being relieved and the temperature being practically normal. Both ears were

kept irrigated with a hot carbolized solution. The patient seemed apparently well. Appetite good and sleep uninterrupted. There was no pain, tenderness or edema over the mastoid. With the exception of the profuse discharge there were no other complicating symptoms.

Smears taken from the discharge showed the presence of both stapylococci and streptococci organisms. To all appearances the case was progressing nicely and healing of the middle ear seemed to be taking a normal course. The only exception to this was the continuous free discharge of mucopus.

On March 9th, four days after the beginning of the middle ear discharge, the patient was seized with an acute arthritis of the finger, knee and ankle joints on both sides. This was accompanied by a sudden rise in temperature 102 F. with intense pain on moving these joints and pain even without movements. Patient was unable to sleep day or night without an anodyne. The usual remedies for this condition were tried without success and after a consultation we decided that there was a toxic absorption from the middle ear indicated by the very profuse discharge and while there were no typical mastoid symptoms it was evident that this profuse discharge could only be coming from the mastoid antrum and cells. The urinary examination showed nothing abnormal. No blood count was made as we did not deem it necessary.

Under the circumstances we decided to open the mastoid. Accordingly on March 12th, one week after the membrana tympani was punctured the right mastoid was operated upon, this being the first ear involved and from which the discharge was most profuse.

A simple mastoidectomy was performed. On entering the mastoid antrum pus was found and a few adjoining cells involved. These were rapidly cleaned out and a free communication made with the middle ear. The wound was closed entirely and a small wick of iodoform gauze placed at the lower end of the wound.

In one week's time all discharge had ceased and the mastoid wound was entirely healed. There was only a slight amelioration in the joint symptoms and the discharge from the left ear was now very profuse. Consequently one week after the first operation the left mastoid was opened and almost identical findings resulted. The wound was closed in the same manner. It took only 20 minutes for each operation.

For a few days there was considerable discharge from the external canal and also from the lower part of the wound. However this rapidly disappeared under treatment and in less than two weeks the mastoid wound was entirely healed and the middle ear was dry. The hearing in both ears was normal. Two days after the operation all arthritic symptoms in the joints disappeared and the patient has had no further trouble. In addition to the double mastoidectomy autogenous vaccines were made from the middle ear discharge and five injections were given in increasing strengths.

As has been mentioned previously it is surprising how few cases of a similar character have been reported. While my tabulated report is perhaps by no means complete the writer has appended an abstract of all the cases which he could find and which will at least aid in further research along this line.

Purulent Pneumococcic Arthritis in Children. Dudgeon and Brausen, Lancet. 2:316, 1903.

Five cases are reported only one of which is related to otitis, Case II.

A boy, 6 years, admitted Mar. 23, 1903. The patient had been ill for three weeks with earache and subsequent otorrhea, when on Mar. 16 he complained of swelling in the left knee joint, followed on the next day by similar affections of the right hip and wrist. There was no history of rigor, convulsion, or any other symptom of note. The patient on admission appeared to be very ill and lay curled up in bed with all his joints flexed. He had a troublesome dry cough and was slightly delirious, though his fever was moderate.

Nothing abnormal was discovered in the lungs or heart. The left knee joint was swollen, red, hot, shiny and fluctuating, while the right hip and wrist appeared to be in a less advanced state of inflamation, and this in the latter seemed not to involve the joint but to lie superficial to it. Urine was normal. The knee joint was opened and a quantity of thin pus and flakes of fibrin were evacuated and a similar result attended the opening of the hip joint. Little benefit resulted from the operation though the wounds progressed favorably. Fever continued high at night with morning remissions and with gradual loss of strength, but without specific complications; the child died from asthenia 20 days after operation. No post mortem was allowed.

* * *

In this series of five cases, the exciting cause of the arthritis in three was found to be "pneumonia" "bronchitis" and "otorrhea." A table of Netter, quoted by Muir and Ritchie, gives otitis media as a source of infection (arthritis) in children in 29 out of 46 cases collected, broncho-pneumonia in 12 meningitis in two, pneumonia in one, pericarditis in one, and pleurisy in one. No case of primary arthritis occurs in the list.

* * *

Mastoid Cases at Camp Sheridan, Christian R. Holmes. Am. Otol., Rhinol. and Laryngol. 28:1, 1919.

In a series of 40 cases reported of mastoid diseases with complications one is given as double.

Case 34: Evans—double otitis media. Simple mastoidectomy left side, March 14. Temperature 106.5 (rectal) at operation. Fell to normal and remained so for about a week, when inflammation developed in left knee, with a moderate rise of temperature. Capt. Sturgis aspirated. Continued mucopurulent fluid. April 3 it became necessary to make a simple mastoid operation on the other ear, at which time the knee was aspirated again. Fluid dark and cloudy.

* * *

Infective Arthritis Complicating Otitis Media.. W. P. Eagleton, Arch. Otol. 34: 483, 1905.

Case 11. Male, aged twenty four. Previous history negative. Never suffered from rheumatism. Otitis media suppurative following coryza. Paracentesis made under nitrous oxide. Very profuse discharge. Temperature 101—103° F. One week later pain in right knee, accompanied by only slight swelling. The next day, the left knee was similarly affected, and later both ankles, accompanying which a distinct pericardial or possibly an endocardial murmur could be heard, which was not present on the previous day. Gradual recovery.

In true infective arthritis and that complicating osteomyelitis, the mortality is very high, while the mobility of the joint is almost sure to be seriously impaired if not altogether lost. The author found only 79 cases reported of pneumococcic arthritis of which 60% (48) died.

Case of Mastoiditis Complicated by Suppurative Arthritis of the Shoulder. Erskine. Ann. Otol. Rhinol. and Laryngol. 25: 1035, 1916.

The mastoiditis developed after an attack of influenza. The patient had been confined to bed for a week and then after having had an earache for a day and a half the ear was examined and a hemorrhagic bleb was found on the drum membrane. The drum was incised and drained. The patient improved for five days, then again suffered with earache. After a week, a culture showed streptococcus brevis. A mastoid operation was performed about which there was nothing unusual. The man was over six feet tall, below par, weighed 147 pounds.

On coming out of the anesthetic the man complained of pain in the shoulder of the opposite side; he declared that it felt as if it were dislocated. There was tenderness over the front of the shoulder and the movements were painful. The next day it showed redness and three days later, when the joint was opened the arm was very much swollen. The temperature was 102 to 103° F. The man had profuse perspiration but no chills and no drop in temperature. The blood culture was negative. A general surgeon opened the joint and found three ounces of serous pus. The culture from the mastoid showed streptococcus, the culture from the pus of the shoulder showed streptococcus, as did the pus from the ear before the mastoid. Immediately after the incision of the shoulder joint the temperature went down. Subsequent course uneventful to recovery.

Dr. Opdyke told of the case of a boy with a mastoid in the right ear and arthritis in the elbow, knee and ankle joint. A year later he had a mastoid on the other side and the same involvement of the other ankle, knee and opposite elbow. The ankle, knee and elbow were not opened in either instance, though they were involved for a long time.

* * *

A Case of Pneumococcic Pyemia with Recovery: A. T. Davis and W. L. Brown; Lancet 2:1017-1020; London October 8, 1904.

1. A table is given of 39 fatal cases of multiple pneumococcal infection occurring at St. Bartholomew's Hospital, London, between January 1894 and August, 1904, where the lesions were bacteriologically proved to be due to the pneumococcus alone, the probable course of events is indicated and the pathologic report appended in each case.

2. Case 35 listed—girl thirteen years old—shows the primary exciting cause as otitic media; complications arising are infective mitral endocarditis, rupture of tendinous cords, splenic and renal infarcts, suppurative arthritis of the right shoulder—and wrist joints; pneumococcus bacillus was cultivated from vegetations; no detailed history is given not any nearer date than that the case came up "in the last ten years."

* * *

Relation of Infective Foci to Rheumatoid Arthritis: J. Lindsay; F. S. P. Strangenays, ed. Bull. Com. f. the study of Spec. Dis. 2, No. 3: 106-116, Cambridge, Engl. 1908.

1. Report is based on 172 cases of rheumatoid arthritis at Royal Mineral Water Hospital, Bath. Engl.

2. The 172 cases included 138 females, 34 males; information on definite source of infection preceding onset of disease obtained in 73 females, 15 males.

3. Otorrhea present in 7 out of 138 females, in 3 out of 33 males.

Case Reports on Otorrhea:

Case 48; married printer; aged 43; onset of rheumatoid arthritis 10 years ago; complicated by spondylitis deformans; has had discharge from left ear for many years.

Case 55; married woman; age 57; onset 4 years ago; prior to this abscess in left ear from picking ear with hairpin; intermittent ear discharge since.

Case 101; married woman; age 31, double otorrhea, very offensive discharge; present in both ears from childhood.

Case 104; girl; age 12; discharge from both ears since childhood; onset at age of 7, slight discharge from left ear on admission to hospital.

Case 108; boy; age 11; onset 12 months ago; intermittent discharge from right ear since age of 6 or 7, discharge recurs apparently on exposure to chills.

* * *

Case Histories of Pneumococcal Osteomyelitis: Blecher; Deut. Ztschr. f. Chir. 48, No. 4:413-417; Leipzig. June 30, 1898.

Case History:

1. Boy; age 12, spontaneous left otitis media purulenta beginning Nov. 2nd, 1897, subsiding two days later; Nov. 5 painful swelling in right femur; rigor and general malaise; admitted to surgical clinic Nov. 8th.

2. Clinical findings—well developed boy; temperature 39.9 degrees; pulse 110; skin over right femur reddened; knee much swollen; lower third of femur painful to touch; deep effusion into knee joint; left ear dry; defect of drum membrane; tympanic membrane moist and shiny.

3. Operative findings—medial and lateral incisions made on femur; 2 isolated abscesses found below detached femoral periosteum, both communicate with the upper recess; bilateral arthrotomy of knee joint; drainage of pus; iodine gauze plug; dry bandage.

4. Microscopic findings—pure cultures of Fraenkell—Weichselbaum's capsulated diplococcus were obtained both with pus drained from wound and with secretion from left ear; secretion from nose contained the same bacillus, but no pure culture could be obtained; otitis media was therefore primary cause of arthritis.

5. Post-operative course—fever persisted until end of November though no new focus of infection or any retaining of pus could be demonstrated; pus formation decreased gradually; in December temperature became normal; granulations formed and on Jan. 15, 1898, the wounds were well healed; after recovery there was still marked restricted motion of the knee joint and a moderately painful swelling of the lower femoral end.

* * *

Acute Otitis Media As An Early Symptom in Acute Rheumatoid Polyarthritis: C. Wolf; Arch. f. Ohrenhlk; 41, Nos. 3 & 4; 213-216; Leipz. Dec. 30, 1896.

Case of Bilateral Otitis Media and Acute Polyarthritis:

Male; 50 years old; left subacute otitis media and diminished hearing June 8, 1888; 3 days later slimy, flaky secretion, hearing improved, on day following right otitis media; drum membrane incised and bloody, serous, later slimy, flaky secretion drained; pain relieved; evening of fifth day rise in temperature to 38.6 degree C; temperature on sixth day 39.2 degree C; headache, pain in limbs, loss of appetite and general strength, on seventh day symptoms of acute rheumatism, painful swelling of both metatarsal joints; the disease progressed, many other joints becoming involved in succession; endocarditis was an added complication; by the fourth week both tympanic cavities healed but still showed slight thickening of the epithelial layers; hearing diminished to about half of normal acuteness; 1½ years later death occurred from endocarditis.

Case of Right Otitis Media and Inflammatory Polyarthritis:

Male; aged 18, slight angina April 1, 1896, four days later right otitis media; livid, moist, bulging drum membrane; hearing almost gone; bone conduction to right increased; Rinne positive; temperature 38.6 degree C; April 5, incision of drum membrane, draining bloody, serous secretion; April 9 no pain, no secretion, improved hearing; on 8th day incision healed, slight temperature, general health unimproved, continued constipation, anorexia, sudden rise in evening temperature to 39.5 degree C., severe pain in various body portions particularly in neck and back, no proof of any other diseased organ established; on 9th day marked swelling of right knee joint; successively most other joints became involved, finally the cervical vertebral articulation also; in the course of inflammatory polyarthritis considerable pericardial exudate developed; later exudation developed in right pleural cavity; several attacks of cardiac weakness; irregular pulse up to 124 per minute; emaciation; gradual improvement after 9th week; complete recovery after a prolonged stay in the country; re-examination October 23, 1896; tendinous thickening of right drum membrane,

handle of malleus and short process of the malleus more prominent than in left ear, hearing in right ear 6m. for whispered speech, in right ear 12 ÷ m; Nov. 6, 1896, recurrence of inflammatory polyarthritis in right shoulder joint and exudate in right pleural cavity, no preceding angina or otitis media; recovery on 10th day.

* * *

A Case of Acute Articular Rheumatism Complicating Acute Otitis Media: Joseph Popper, Med. Rec. 99, 270, Feb. 12, 1921.

Report of a case of acute articular rheumatism following acute purulent otitis media and considered interesting, both on account of its rarity and because of the fact that this condition may be so easily confused with the bacteremic condition following severe cases of middle ear disease usually complicated by mastoiditis or lateral sinus thrombosis.

The patient, male, nine years old, was admitted to the hospital complaining of discharge from the left ear and pain in the right ankle. The child had had all the usual diseases of childhood with good recovery. His tonsils and adenoids had been removed two years ago. The family history was irrelevant. Ten days before admission, he had been put to bed with a temperature of 102 degree F., complaining of headache, running nose and slight, dry cough. Two days later pain developed in the left ear, from which there was a spontaneous discharge of pus that night. The fever ranged between 101 degrees and 104 degrees during the ten days prior to admission. The discharge from the ear became scanty, meanwhile, and on the eighth day of the illness, an otologist enlarged the opening in the ear-drum without, however, increase of the discharge following. On the ninth day, there was pain in the back and on the tenth day, pain in the right ankle. Physical examination showed that the head, eyes, right ear, mastoid, neck, heart, lungs, and abdomen were normal. Spine and reflexes were normal, as were the urine and blood. There was a small amount of mucopus present in the left ear, but the drum was not bulging. Following admission, the patient experienced pain, tenderness and disability in almost all the large joints, and some of the small joints of the hands. By the sixteenth day the temperature, which had been ranging from 101 degrees in the morning to 104 degrees in the afternoon, declined and reached normal on the nineteenth day. With this decline, the symptoms cleared up and the general condition improved remarkably. The patient had been treated expectantly until the blood cultures were reported negative. This fact, together with the absence of chills, led the author to believe the case one of acute articular rheumatism, as seen in childhood. Salicylates were given and in twenty-four hours symptoms were markedy relieved. The author suggests that acute articular rheumatism may be a symptom-complex secondary to some focus of infection, rather than a definite pathological entity.

DISCUSSION.

Dr. Frank R. Spencer, Boulder, Colo., said that reporting these cases was valuable. One sees more cases of middle ear and mastoid foci of infection in children, than in adults.

Dr. Ewing W. Day, Pittsburgh, Pa., said that Dr. Roy had evidently very mild cases of blood stream infection from the mastoid. He had seen 4 cases where there was a typical blood stream infection with hemolytic streptococci, and the disease ran a very severe course.

Dr. Dunbar Roy (closing) said that the highest temperature was 103. The discharge was profuse and he operated to relieve the infection. The result showed the rationale of his judgment for after free drainage the symptoms subsided, with no signs of sepsis except the general involvement.

THE UPPER RESPIRATORY TRACT IN GRANITE DUST INHALATION.*

By D. C. JARVIS, M. D., Barre, Vermont.

Vermont having the largest granite center in the world, the opportunity to study the effects of granite dust inhalation upon the respiratory tract is exceptional. A permanent residence rules out the time factor and makes it possible to carry the work to any conclusion thought advisable. Observing the upper respiratory tract of granite cutters for the past twelve years one's attention could not fail to be focussed sooner or later upon their difficulties, and in 1917 a definite effort was made to observe in detail the condition these men manifested.

Introduction. The accumulation of cases was slow and the advent of The National Tuberculosis Association in the field during 1919 was welcomed. Through its Committee on Mortality from Tuberculosis in the Dusty Trades an investigation of the granite industry was started because it was thought that the granite industry of all the dusty trades would show the highest mortality from tuberculosis. The investigation was divided into three phases: A Statistical Inquiry, A Mechanical Process Inquiry and the Medical Examinations. The granite cutters have shown a remarkable spirit of co-operation. They did not appear because of any illness but came up for examination in routine order by previous arrangement. This previous arrangement consisted in taking the subject up with the Executive Committee of the local Union, they in turn calling a meeting of the men for a consideration of the subject and action. Many of them had never had occasion to consult a physician before. They were all able to work and considered themselves in good health. At this time it is possible to report on the examination of five hundred granite cutters.

Review of the Literature. While numerous references are found in the literature to dust inhalation in general and some forms in particular from Ramazzini's Article in 1700 down to the present time, one fails to find a record of a definite study

*Candidate's Thesis Recommended for Publication by the Council.

of the effects upon the upper respiratory tract of granite dust inhalation. The term "stone cutters" is found but with no particular effort to separate granite cutters from marble workers and stone masons. The General Report of The Miners Phthisis Prevention Committee of South Africa, published under date of March 15, 1916, comes the nearest to the present work, but their similarity lies in the composition of the stone and not in the manner of handling it as the South African process I understand to be a blasting one while the Vermont process is in finishing the stone for monumental purposes, the stone being worn down by means of a pneumatic tool. The work in Vermont represents the first work of The Committee On Mortality from Tuberculosis in The Dusty Trades. The only other investigation of a dusty trade in this country is represented by the valuable contribution to be found in United States Bulletin No. 85, issued January 1917, comprising a clinical study of 433 cases of Miners Consumption among Zinc Miners in Southwestern Missouri by A. J. Lanza.

Experimental Pathology. Dr. L. U. Gardner of the Saranac Laboratory is in charge of the experimental pathology connected with the investigation, and the reader is referred to his article in the December 1920 number of The American Review of Tuberculosis for the preliminary details of this phase of the work. One specimen from the upper respiratory tract examined by Dr. Gardner was reported as follows. "Examination of tissue from granite cutter's nose of Sept. 25th shows a mucous polyp composed of a mucous connective tissue stroma covered with columnar epithelium on the surface. Mass contains frequent gland acini and is in places infiltrated with plasma cells. No trace of granite dust nor any reaction suggesting this irritant."

Evidence on which Report is Based. The economic phase was so important that attention was paid to small details such as the presence of a moustache, its growth as to luxuriance, the length of time it had been worn, and the amount of hair in the nose; the condition of the septa; the turbinates; the presence of lymphoid tissue upon the posterior pharyngeal wall; the condition of the tonsils, teeth and the larynx. From the observations necessary in accumulating this evidence the deductions and conclusions made in this thesis have been derived.

Composition of Granite Represented by this Study.

Aluminum Oxid	15.38
Aluminum Oxide	15.38
Iron Oxide	2.65
Magnesia Oxide	Trace
Calcium Oxide	1.76
Sodium Oxide	5.38
Potassium Oxide	4.31
Manganese	Trace
Loss in Ignition, Co2 and moisture	1.02
	100.06

According to Gardner when examined under the microscope the unscreened dust may be roughly divided into large and small particles. The former, which could not possibly be concerned in an inhalation experiment show an average size of 82 x 84 microns. The smaller particles average 4.3 x 6.3 microns by actual measurement. In shape the particles may vary greatly. Some are flattened polygonal masses with sharply cut edges and corners. Others are rod shaped, often slightly curved with sharply horizontal or square cut ends. Frequent vegetable cells and fibers are noted. Samples of dust resulting from the use of the pneumatic tool have been examined by means of a small giant magnet made by the Victor X-Ray Corporation and considerable steel has been extracted from a given quanity by means of the magnet; 2 drams of granite dust yielding 2 grains of steel. From this experiment it will be seen that we are dealing with two kinds of dust that from the stone being worked and that from the tool used in working it.

Present Problem. In 1915 we found 2050 granite cutters working while in 1919 we found only 1240. The question suggesting itself whether this condition was peculiar to Vermont alone, the total number of cutters for the United States was investigated and it was found that there was a corresponding diminution in the number of cutters throughout the whole country. The statistician returned the report that an analysis of the death certificates for the past twenty years indicated that 85% of the cutters died from tuberculosis. The present study was undertaken to determine if possible the part played by the nose and throat in the developing of the clinical condition. Realizing that a study of the industry reveals men who have inhaled

granite dust with impunity over periods ranging from thirty to fifty-nine years, and other men who are punished severely by granite dust inhalation even when less than fifteen years represents their period of exposure, one wonders why. Why do some men suffer severely from the effects of dust inhalation, and why do others escape so easily.

The Occupation in Itself. Because an occupation is dusty apparently one has no right to jump to the conclusion that affections of the respiratory tract are prevalent to a large degree. Also if an individual working in a dusty trade develops an affection of the respiratory tract one apparently has no right to jump to the conclusion that dust inhalation was its cause. There are other factors such as hygienic conditions to be considered, and while a close study of granite dust inhalation reveals the fact that a certain proportion of the men have an affection of the respiratory tract superimposed, still one who works among these men daily cannot help but feel that a vicious circle is developed; that the inhalation of granite dust in itself is not a producer of a serious respiratory disease but rather produces other conditions of the respiratory tract less serious, such as frequent colds, bronchitis and pneumonia, which in turn prepare the respiratory tract for the superimposing of a tuberculous process.

Suitable Soil. As one works longer among these men there comes a feeling that certain of them represent suitable soil for the early manifestation of the injurious effects of granite dust inhalation. Knowing that all men were not safe in the industry, and that some broke down earlier than others, it became necessary from an economic standpoint to ascertain if possible the type of individual who would not last long in the industry under present working conditions, and suggest that he take up some other trade, for while the period of apprenticeship is three years it takes between eight and nine years to bring a cutter to a final degree of excellence. While these men have been studied during the past twelve years it is only during the past four years that an effort has been made to discover the type of individual who might be accepted as an apprentice, and the type representing a hazard, which if accepted would soon break down in the industry and represent an economic loss to both industry and state. It seemed advisable to first examine the survivors in the industry to ascertain if possible the individual characteristics they possessed and their mode of life and so cutters who

had inhaled granite dust as long as fifty-nine years were examined and detailed data kept of their condition. As stated before, the economic phase was so important that attention was paid to small details. Cutters who had broken down were examined and several hundred cutters who were working all the time, endeavoring to find the type of individual who would last reasonably long in the industry. As a result of all this work one is able to tell the type of individual representing a hazard, and keep him out of the industry and select the individual who would do well. It was found that the upper respiratory tract held the key to the situation and that an individual was a good risk in proportion to the absence of lymphoid tissue in this region. An apprentice with normal turbinates, a smooth posterior pharyngeal wall, tonsils small in size, no evidence externally of enlarged lymph glands, represented a desirable type and one who under ordinary conditions would last in the industry. On the other hand if a young man 20 years of age showed hypertrophic rhinitis, enlarged tonsils and lymphoid nodules on the posterior pharyngeal wall he should keep away from the industry as he will break down fairly early under present working conditions without dust removing devices. On the basis of suitable soil it was found that the different nationalities lined up in a definite manner. In order of excellence they were found to line up somewhat as follows: Italians, Americans, English, Spaniards, Scotch, Swedes, Norwegians, Danes, French and Irish. You will notice that there is a geographical distribution of these races, many of those being least affected by the dust, such as the Italians, living in Southern Europe, and many of those along the last of the list living in Northern Europe. They may be also classified on the basis of those living in dusty countries and those living in countries not so dusty. It cannot be denied that in the granite industry at least the Irish are punished severely, and it is a question whether they should be in a dusty trade, certainly not in the granite industry, as they represent a hazard under present working conditions, and should not be accepted as apprentices.

Suitable Preparation of Soil. As a result of granite dust inhalation there occurs an adaptation to occupation of the upper respiratory tract which one is able to estimate in various ways. If one draws a pencil point across the posterior pharyngeal wall of a cutter he will not know by the feeling that anything has

touched his pharyngeal wall. One may hold a laryngeal mirror against the soft palate indefinitely, and the cutter states, that he feels nothing in his throat. The ordinary gargles are tasteless to him, and it is only by using them very hot and all throat applications in increased strength that one is able to secure results. The adaptation to occupation is best studied in connection with the eye for here one is able to remove foreign bodies from the cornea without the aid of cocaine, and these may be present without the usual accompanying signs of irritation. Small pieces of granite 2 to 3 millimeters in length have been observed floating about in the conjunctival sac without the cutter being aware of the presence of a foreign body. This adaptation to occupation apparently is a defense whereby dust inhalation is tolerated but while it acts as a defense during continued work it acts as a detriment when absence from work is necessary for any great length of time, because in proportion to the length of time a cutter remains away from work in like proportion does he loose his adaptation to occupation. It is a well known fact that if a cutter is absent from work one month on his return for the first three or four days he expectorates a great deal of dust, the same coming up in little round balls, which are moist on the outside but when rolled between the thumb and finger the center of these little granite balls is found to be dry. With the passing of three or four days he ceases raising, adaptation to occupation having occurred again. With longer periods of idleness this adaptation to occupation is not so easily regained. Cutters themselves have discovered that something happens as a result of these periods of idleness brought on by strikes, lockouts, or poor business because they have observed and so state that a resumption of work following one of these periods of idleness causes the breakdown of many men. Granite dust inhalation in itself is not the direct cause of tuberculosis being superimposed, but it is the suitable preparation of the soil occurring when a cutter is endeavoring to acquire again his adaptation to occupation that assists in spelling his downfall. Here then we have a vicious circle, granite dust inhalation requiring adaptation to occupation of the respiratory tract, absence from occupation causing him to lose this adaptation and a reentry into the industry producing irritation of the respiratory tract while he is endeavoring to regain his lost adaptation, this last effort bringing about a suitable preparation

of the soil. With two hundred dusty trades listed in the United States one wonders how much of a factor his adaptation lost and regained many times during a worker's occupational life is in the production of disease of the respiratory tract. Certain it is that if a cutter absents himself five years he may return if his initial exposure has been less than ten years, but any cutter who has been absent from the industry two years should think twice before reentering because the difficulty in regaining adaptation to occupation represents a handicap too great for many to overcome, and suitable preparation of the soil takes place with the developing of a tuberculous process as a result.

Mouth Breathing. According to the experience of Watt Irving Johnson and Stewart in the South African investigation the mouth breather is more apt to suffer from the effects of granite dust inhalation than one who employs nasal breathing. Particular interest has been taken in this phase of this subject and Roentgen ray films of cutters showing mouth breathing were separated from those showing nasal breathing, and a study was made comparing them with other films of the same exposure period, with the result that one cannot help but observe that the films belonging to those breathing through the mouth show a lesser lung lesion than those breathing through the nose. The explanation is that the usual curve of the inspired air is broken by mouth breathing, and the dust impinging upon the posterior pharyngeal wall is expectorated as soon as the accumulation warrants or is swallowed with the result that very little reaches the trachea and the lungs are consequently spared.

The Nose. While the septa showed deviation in a number of cases, the turbinates did not give evidence of hypertrophy, apparently adaptation to occupation taking place readily. It would seem from this study that inhalation of a dust as irritating as granite does not exert a marked influence upon the size of the turbinates, the causal factors producing hypertrophic rhinitis coming from within rather than without the body. The wearing of a moustache, or the amount of hair in each vestibule did not seem to exert an influence upon the appearance of the Roentgen ray films.

Diet in Relation to Lymphoid Tissue. The diet was investigated and it was possible to study ten nationalities in this respect with the result that the evidence showed that the more greasy

the diet the less amount of lymphoid tissue there seemed to be in the upper respiratory tract.

Practical Application of the Evidence Gained from this Study.

First. In view of the fact that adaptation of the respiratory tract to occupation occurs in dusty trades, in event of bronchoscopy becoming necessary the patient's occupational history should assist in deciding upon general, local or absence of anesthesia. It would seem that in a granite cutter at least the passage of the bronchoscope might be accomplished without the aid of an anesthetic.

Second. An individual it would seem is susceptible to affections of the upper respiratory tract in proportion to the amount of lymphoid tissue present.

Third. In event of an individual contracting tuberculosis assistance in its prognosis may be gained by studying the lymphoid tissue of the upper respiratory tract, the prognosis being better in proportion to the absence of lymphoid activity in this region.

Summary. The evidence tends to show that there exists in certain individuals suitable soil for the development of respiratory diseases.

That in the worker in a dusty trade adaptation of the respiratory tract lost and regained many times often acts as suitable preparation for developing a serious respiratory condition.

Mouth breathing is not as injurious as nasal respiration in granite dust inhalation.

An irritant such as granite dust from without does not seem to produce an increase in size of lymphoid tissue in the upper respiratory tract. An inquiry as to the occupational history may assist in deciding the method of anesthesia when contemplating work upon the respiratory tract.

The strength of local applications to the nose and throat should vary with the respiratory environment of the individual.

REFLEXES FROM THE RESPIRATORY TRACT.*

By HOLMES C. JACKSON, Professor of Physiology, University and Bellevue Hospital Medical College, New York University.

The importance of the respiratory tract in guaranteeing to the lungs proper ventilation, renders it necessary that every part of the body should be rapidly linked up with the lungs and air passages by reflex control. Johannes Müller recognized this when he said "irritation of any mucous membrane of the body is the most frequent cause of reflex manifestations of the respiratory system". Likewise for perfect coordination of the body these air passages and the lungs should be brought reflexly into close accord in the reverse direction with other constituent parts of the body. And it is certainly true that the various portions of the respiratory tract are reflexly related to one another and to the contiguously situated organs, the eye and ear. All reflexes function as a part of the protective machinery of the body. In fact the reflexes which we plan to discuss are those which in the main serve the purpose of guarding the respiratory tract from injury. The appearance of dangerous conditions in the environmental air such as smoke, ammonia, dust, etc., sets up as soon as these substances enter the nasal atrium, reflexes in the nose itself, in the eyes, in the pharynx and larynx whose purpose it is to remove the noxious matter. Also the development of abnormalities of growth or bacterial infection acts as an irritant and sets up protective reflexes.

Reflexes from the Nose.

It will perhaps be advantageous for the sake of proper understanding if we quickly sketch the possible types of reflexes which may be involved in the further discussion. The neural structure which underlies the axon reflex represents the most primitive form of nerve connection. Reflex action over such a pathway is possible without a central nervous system. Two forms of neural architecture for axon reflexes have been described, the first in which only one neuron is involved and the second where two neurons synapse in an autonomic ganglion The first and simplest form is that in which the afferent axon at the periphery gives off collaterals to the skin as well as to

*Read by invitation.

blood vessels in the same cutaneous area. A neural structure of this form is the basis for the hyperemia which occurs when mustard oil is placed on the conjunctiva. This is not a direct action of the irritant since cocain inhibits it. The reaction is therefore brought about by nerve pathways; however, the central nervous system plays no part since section of the spinal roots supplying the conjunctival area, does not preclude the reaction. It has been shown that stimulation of the peripheral end of a severed posterior root will bring about a vaso-dilation of the area enervated by that root. This does not prove that posterior roots carry efferent fibres but as Bayliss supposes, the neurons involved in the reaction are of the ordinary afferent type capable of conveying not only sensory impulses into the cord from the blood vessels but also able to bring about vaso-dilation as well. This would seem to indicate that if impulses, sensory in character, originating at the surface of the body can spread through out the neuron and all its collaterals, and if one of these collaterals were in relation with the blood vessel of that area, then stimulation of the surface would bring about vaso-motor changes without the intervention of the central nervous system. The reaction is one mediated within a single neuron and is therefore called an axon reflex, one collateral acting afferently and another efferently. The second type of axon reflexes involves a post ganglionic neuron passing through an autonomic ganglion and giving off, on its way to the periphery, a collateral which synapses within the ganglion with another postganglionic cell body. Stimulation of the former neuron at the surface of the body would, through its collateral synapse discharge the second postganglionic neuron and thereby initiate a peripheral effect.

Reflexes involving the central nervous system as centers are either purely visceral, i. e. autonomic, or mixed somatic and visceral. In the former type the afferent neuron is a sensory visceral cell such as the taste fibres and the efferent neuron an autonomic, as for example the secretory fibres to the submaxillary gland. The neural architecture of the mixed reflex would include an afferent visceral pathway like that from the nose to the pons and an efferent somatic fibre conducting from the center to the respiratory muscles, the reflex involved being a sneeze or cough. This latter is the usual sensori-motor reflex reaction involving striated muscle.

Anatomy of the Nasal Reflexes.

The nasal mucosa receives its visceral afferent supply from the trigeminal sensory divisions, the ophthalmic and maxillary, principally the latter. These fibres would constitute the afferent arc in reflexes from the nasal cavity. Impulses originating in the septal mucosa as the result of some irritation in this region would pass via the nasopalatine nerve to and through the sphenopalatine ganglion and then to the maxillary division of the trigeminal by means of the sphenopalatine nerves. Following the maxillary nerve to the trigeminal ganglion (the Gasserian) the impulses enter the pons by means of the sensory root of the trigeminal nerve. From other portions of the septum impulses would be conducted via the internal division of the naso-ciliary branch of the ophthalmic nerve to the trigeminal ganglion and from thence to the pons as before. From the lateral wall impulses traveling over the nasal and palatine nerves reach the sphenopalatine ganglion. The pathway from this point would correspond to that outlined for the nerves of the septum. It must be remembered that the sphenopalatine ganglion belongs to the autonomic system. Passing through it are the afferent fibres just mentioned together with efferent autonomic neurons, cranial and sympathetic. The cranial preganglionic fibres, originating in the motor nucleus of the seventh nerve in the pons, leave the nerve as it passes through the petrous portion of the temporal bone as the great superficial petrosal which joins the vidian nerve to reach the sphenopalatine ganglion. These preganglionic fibres synapse here with post ganglionic cell bodies the axons of which are carried in the palatine nerves to supply the blood vessels and mucous glands of the nasal mucous lining. The sympathetic (cervico-thoracic autonomic) postganglionic neurons arise in the superior cervical ganglion. They pass to the internal carotid plexus and from thence travel via the great deep petrosal nerve and vidian nerve to the sphenopalatine ganglion; from this they are distributed through the palatine nerves. It is probable that the post ganglionic fibres from the sympathetic autonomics in passing to the septal mucosa through the sphenopalatine ganglion give off co'laterals which synapse with the cells of origin of the post ganglionic fibres of the cerebral autonomics. This gives us the neuron architecture of an axon reflex of the second type.

Central Connections.

We have just seen that the trigeminal nerve carries afferent conducting fibres which pass into the pons. Here they divide into an ascending branch terminating in the main sensory nucleus; the descending collateral runs through the spinal tract and ends in the nucleus of the spinal tract of the trigeminal nerve. This group of cells receiving some fibres from the glossopharyngeal and vagus nerves becomes continuous in the spinal cord with the substantia gelatinosa Rolandi. There is consequently a continuous column of grey matter extending from the sacral region in the cord to the pons and capable of acting as a receiving station from the afferent fibres of the trigeminal and a discharging center to practically all segments of the cord. From the cells of the main sensory and spinal nuclei of the trigeminal nerve, fibres arise which enter the reticular formation and are grouped into longitudinal bundles from which collaterals are given off to the motor nuclei of the brain stem, thus establishing reflex connections with the efferent fibres of the cranial nerves. Ascending fibres enter the main sensory tract of the opposite side which ends in the thalamus, and synapse with sensory radiations to the cortex. By this means sensation becomes a component part of certain reflexes such as the sneeze and cough.

It thus becomes apparent that with the possibility of such wide spread interneural connection in the pons between the sensory afferent somatic and visceral fibres on the one hand and the motor nuclei of origin of all the cranial nerves carrying motor fibres (somatic and visceral) as well as those in the cord via the substantia gelatinosa on the other, reflexes to practically all parts of the body may be initiated from the nose.

General Functions of the Nose.

We must pause just a moment to consider the rationale and the function of various parts of the respiratory passages. The nose functions as the atrium of the air passages. Its olfactory epithelium was developed primarily as the receptor surface or sensifacient area of a protective reflex by means of which we might with the aid of the olfactory sense tract and cortical center, become conscious of the proximity of dangerous gases or air particles. The nasal mucosa not concerned with olfaction has for its purpose the secretion of fluid which raises the water vapor tension of the air inhaled until it becomes practically saturated. The massive blood supply to the mucosa allows of

ample heat radiation by means of which the temperature of the inspired air becomes that of the body. The hairs or vibrissae of the vestibule act as filters for large particles of dust and bacteria carried in from the inspired air. The ciliated epithelium completes the task of removal of the bacteria by assisting the fluid, secreted by the eyes and nose, to flush the surfaces of the cavity. It has been said that the nasal mucous lining may show bactericidal properties. Air reaching the nasopharynx should be practically sterile. In mouth breathers removal of bacteria occurs only in part, by the moist surfaces of the mouth and pharynx. Since the nose is normally the main point of entry of gaseous substances to the body, it is quite understandable that it should be reflexly coordinated with all parts of the respiratory tract and of the body as a whole.

It may be interesting in connection with the thought of the protective function of the nose to recollect the reaction which occurs as an acute rhinitis, a prodromal symptom in so many infectious diseases, i. e. influenza, measles, whooping cough, scarlet fever, diphtheria and erysipelas. The gastro-intestinal tract reflexly attempts to rid or purge itself of poisonous substances by vomiting and diarrhea. Apparently in an analogous way following an infection which usually enters via the respiratory tract, the body sets up reflex reactions which would serve to remove the source of infection if it happened to be nasal in character. The vasomotor dilation succeeded by the profuse discharge which is at first watery then mucous and only later becomes mucopurulent, must be considered as a protective flushing mechanism. That this coryza-producing function of the nose is controlled by reflexes from the surface of the body has been shown experimentally. It is possible to alter the blood supply to the nasal mucosa and thereby the temperature of the nasal cavity by changing the environmental temperature affecting the cutaneous surface of the body. If the temperature of the outside air be lowered, the heating function of the nose is increased by a vaso-dilation of the nasal mucosa. This increase in blood supply is sufficient to materially augment the thickness of the nasal mucosa and to produce a profuse mucous secretion. Even normally the hyperemia may be sufficient to obliterate the lumen between the turbinate and the septum. This bringing together of two surfaces acts as a stimulus sufficient to originate a reflex such as a sneeze so common in these conditions.

Intranasal Reflexes.

Francois Franck in 1889 stimulated with an electric current the edge of the middle and inferior turbinates and obtained reflex hydrorrhea, thus proving the possibility of bringing about intranasal vasomotor reflexes from irritation of the mucosa. The axon reflexes finds its exemplification in the hyperemia and rhinitis which are present and quite common amongst furriers, millers, workers in saw mills and with tobacco, iodine and bromine. The irritants present in the air places where these trades are conducted, initiate the reflex vasomotor response.

Mechanical pressure might be mentioned as one other cause of vasomotor changes from the nasal mucosa. The turbinates normally are in rather close proximity to the septum. Not more than ⅛ to ¼ of an inch of air space intervenes. The air passing through this channel takes up moisture. The drying in itself acts as a stimulus, but the narrower the interspace as in deflected septa, bony and cartilaginous outgrowths, thickening of the mucosa of the turbinate or septum, and polypous growths from the turbinates, the more rapidly the air moves, in fact in some instances sufficient contact to cause irritation may be established between septum and turbinate; this stimulates the sensory nerve endings in the mucosa and produces reflex effects the more prominent of which are sneezing and coughing. These abnormalities may not in themselves originate the reflexes but only in virtue of the hyperemia which they produce. Persistent sneezing with watery discharge is held to be symptomatic of disturbed septal turbinal relation.

Reflexes Initiated from the Nose with Effector Surface Elsewhere in the Body.

Respiration is markedly affected by nasal conditions. Cold water on the skin of the nose as well as sudden chilling of the nasal mucosa inhibits respiration completely and in whatever state of the respiratory cycle the stimulus may be applied. Diving animals show this reflex to best advantage. Strong irritation such as ammonia fumes likewise causes stoppage of breathing, while the electric current markedly auguments the rate and depth of respiration. Tickling of the mucosa or the pulling out of nasal hairs will cause reflex sneezing. These are protective reactions to prohibit the entrance of injurious substances into the respiratory tract. Hazelhurst has described a case with frequent attacks of sneezing always associated with mastication and ingestion of foods. The symptoms disappeared

following removal of a septal ledge which pressed against the inferior turbinate actually indenting it. John Mackenzie mentions certain sensitive areas from which it is particularly easy to elicit a reflex response. These areas are the mucous membrane covering the middle and inferior turbinates and posterior half of the septum. Jacobsen's tubercle lies at the anterior end of the middle turbinate. It will be noticed that these areas embrace those portions of the nasal surface devoted to turbinates which lie in intimate relation with the septum and in which we find the large venous plexuses. This large blood supply would account in part for the normal heightened irritability of these areas.

That the hyperemia is an important feature in the origin of the reflexes seems to be proven from the fact that in atrophic rhinitis, reflexes are not prevalent, although the accumulation of crust may become an exciting cause.

Many observers speak of reflex cough from nasal abnormalities such as polyps, hypertrophic rhinitis, septal spurs and deflections and irritations of the anterior and posterior extremities of the inferior turbinates and inner surfaces of the middle turbinates, so-called cough spots of Francis. It is evident that these conditions are similar to those that produce hyperemia so that the cough which results in some cases is merely an extension of the reflex arc to an area extra nasal. It is not possible to give a reason why these pathological conditions do not always act as excitants to the cough reflex. One might imagine a hyperirritable center as in some neurotic condition, or some abnormal condition of the larynx, or both nose and larynx together might serve as points of somewhat low threshold, neither of which alone is capable of eliciting the reflex cough but both functioning synergetically, able to discharge it.

The possibility of nasal asthma must not be overlooked. The reflex relationship between the nose and bronchial musculature was emphasized by Lazarus who repeating Francois Franck's work, stimulated the nasal mucosa with electrical currents and obtained contraction of the muscular coats of the bronchioles as evidenced by increased intrabronchial pressure. Asthmogenic points, acording to Brugelmann, are small hyperesthetic areas in the region of the middle and inferior turbinates, of different color than the rest of the mucosa. Brodie and Dixon caused a spasm of the bronchial musculature by stimuli applied to the septum. When patients give a history of attacks of

sneezing. Lorie advises that the nose be examined as a possible seat of the asthma. Cures of asthma are reported by many authors when nasal abnormalities are corrected. The conclusion to be drawn from these statements should be that in asthma refractory to treatment based upon anaphylaxis, the nose might well be explored.

Mention in the literature has been made of cases of excessive salivation cured by nasal treatment. Whether the salivation was the manifestation of some unrecognized central lesion or whether the reflex was direct, it is difficult to decide. Anatomically it is possible. The same train of critical thought must accompany the following statements of like nature. Richet reports tic doloreaux cured by removal of a growth from the septum. Sallinger has seen cases of epilepsy apparently due to intranasal disease. Optic abnormalities are deemed to be caused reflexly by nasal conditions together with dysmenorrhea, photophobia, conjunctivitis, asthenopia, epithora glaucoma, and so forth. Many of us may rightly be somewhat sceptical concerning the direct reflex relation between the nasal cause and the syndromic effects just mentioned. It is not my purpose to present briefs in favor or against, but merely to state that anatomically and functionally the reflex effects are possible.

It is always well to be mindful of the fact that in hypersensitive conditions of the central nervous system, disturbances of reflex origin may manifest themselves from small and, under normal circumstances, insignificant causes. The wide spread central connection in the pons of those fibres from the nose through the trigeminal nerve apparently allows relatively slight alterations in the mucosa to show greatly varying effects according to the state of irritability of the central nervous system. Thus for example, sneezing, coughing, glotic and broncheal spasm are more common clinically in neurotic individuals. In fact Bosworth stated that extreme tendencies to intranasal congestion when the hyperemia became pronounced, was indicative of a neurotic temperament.

Finally the subject of nasal misreferred pains must not be overlooked even though they are not strictly reflex phenomena. The nasal mucous membrane is not a place of high pain sensibility. According to Head, impulses originating on such a surface and capable of bringing about painful reaction in the cortex would be misreferred to the cutaneous surface as a place of higher sensibility. However, on account of the great area

of distribution of afferent fibres in the 5th, 7th, 9th and 10th nerves and their close central connection, any low threshold point will bring about a misreference over a wide spread cutaneous area. The pains of trifacial neuralgia in atrophic rhinitis are examples of this, as well as the extensiveness of pains arising from a single tooth. Sluder has spoken of a sphenopalatine syndrome of irritation, pain behind the eye and mastoid which radiates down into the neck and arm to the fingers. It is stated by Thoma that sinus disease causes a painful area referred to the ear, while in simple acute rhinitis the pressure exerted on the sinuses is responsible for the frontal headache through misreference. In certain cases of high deflection of the septum, headaches may appear from negative pressure in the sinuses developed as the result of absorption of air from the closed spaces.

Reflexes from Mouth and Pharynx.

The teeth are supplied by afferent sensory fibres from the dental branch of the trigeminal; the mouth and pharynx from the pharyngeal plexus, palatine branches of the sphenopalatine, glossopharyngeal, vagus, and the sympathetic autonomics. The intimate central connection of these nerves has been previously mentioned. It may be advisable however, to emphasize the fact that the afferent glossopharyngeal fibres entering the medulla along the ventral side of the restiform body help to form the longitudinal tract called the fasciculus solitarius; the lower part of the nucleus of the tractus solitarius is the respiratory center. These central connections are sufficiently complicated to allow the possibilities of reflex pathways from the mouth and pharynx to all parts of the body.

The teeth may act as receptor surfaces for reflexes appearing over widely divergent areas and with greatly dissimilar symptoms. Kirk states that dentitional reflexes may include derangements of speech, hearing or vision. He mentions cases of strabismus among school children as well as chorea, epilepsy, dementia precox and intellectual dullness as the results of improper care of the teeth, and finds that these manifestations disappear after dental treatment. Clark gives as causes of increased dental reflexes, infection of the gums, decay of teeth, impaction, and faulty dental work. From these irritations come reflex neuritis, neuralgia, insomnia and mental disorders.

Concerning the wide area for misreference of pain sensation in the case of the teeth we are all familiar. This requires no

further discussion save merely to suggest that while the teeth may be the originating area for the pain, all facial pains should not be regarded as sufficient evidence for advising teeth removal.

From the pharynx there is elicited the respiratory or breath stopping reflex which occurs when food is on the rear of the tongue preparatory to swallowing. The stoppage of the respiration takes place in any phase of inspiration or expiration. Stimulation of the central cut end of the glossopharyngeal nerve will cause inhibition of the respiration in the passive phase of expiration. The reaction is of course protective and allows the various muscular contractions to supervene in order that the Eustachian and nasal orifices as well as the glottis may be closed so that the food may be shot through the pharynx. The pharyngeal mucosa is particularly responsive to light pressure and stroking, and reflex vomiting or gagging may easily be initiated by placing the finger in the pharynx. The sensitiveness of the mucosa to such treatment varies with different individuals and its absence is well known to be a symptom of tabes. Irritation of another type elicits from the pharynx reflex cough, or hacking. The pharyngeal tonsils especially may function as receptor surfaces for reflexes when they are inflamed through the presence of bacteria or pus. The embedded form is apt to result in more reflex effects because they cannot enlarge greatly without causing local disturbances which act to initiate the reflex. Enlarged lingual tonsils may similarly function as an excitant while pharyngeal reflex cough may also originate as the result of an excess of mucous material from the postnasal pharynx flowing over normal or somewhat inflamed and irritable tonsils. Mackenzie suggests an excessively anteriorly scrolled epiglottis as a provocative of the cough.

Reflexes from the Larynx.

The larynx is supplied by the superior and inferior laryngeal branches of the vagus nerve but the former nerve carries all the sensory or afferent fibres whose cell bodies are in the ganglion nodosum; the central axons entering the pons descend and joining afferent fibres from the glossopharyngeal, form the tractus solitarius and terminate in its nucleus the lower part of which, according to Cajal, is identical with the respiratory center. From the nucleus of the tractus solitarius, descending fibres arise, the tractus solitarius spinalis, which pass in the cord to the primary motor neurons belonging to the phrenic and intercostal muscles. It is thus possible to trace the nervous

pathway over which impulses must pass in the production of the reflex cough from the larynx.

Stimulation of the central cut end of the superior laryngeal nerve will cause the respiration to stop in the phase of passive expiration. If the stimulation is sufficient the bronchioles may also become markedly constricted and cardiac reactions may also appear. This protective response of inhibited respiration can be originated as we have seen from the nose, pharynx and larynx.

In this connection it may be worth while to comment upon the possibilities inherent in the injury under surgical procedures of the superior laryngeal nerve. All are familiar with the dangers especially present when for some reason or other not frequently recognized, a state of hyperirritability of the larynx exists. This may account for some of the disasters of bronchoscopy although it cannot bear the burden of all the fatal results. There are many cases which do not, unfortunately, get into the literature, of deaths occuring after some operative treatment in the neck especially around the thyroid gland or larynx. Two symptoms, usually not appearing in the same case, are prognostic of laryngeal afferent injury. In one, the respiration stops for no evident reason and quite suddenly, with the heart apparently in good condition. Attempts at artifical respiration are unsuccessful, a few gasps may result, the heart during this time remaining fairly normal. Gradually however, in spite of all attempts to start respiration, the heart becomes weaker and death occurs with some asphyxial spasms. Quite evidently a reflex inhibition of the respiratory center has occured, sufficient in degree to repel all attempts to discharge it. In other cases, the heart begins to slow down; breathing may apparently be unchanged. The pulse rate may continue to decrease or the heart may stop suddenly before any respiratory disturbances would seem to warrant such cardiac changes. Here of course, there is no evidence of asphyxial conditions. It would seem that the laryngeal trauma has brought about reflex inhibition of the heart.

Coughing which also may be elicited from a large part of the respiratory passages, must be considered as a remedial as well as a protective process. For diagnosis it possesses the same value as pain, and should be employed similarly. But just as pain has a protective and remedial value so has coughing. Hence the use of antibechics is perhaps contraindicated in many

instances. Chevalier Jackson calls the cough the "watch dog of the lungs" and he reasons that this sentiment protector should not be thoughtlessly put to sleep by narcotics. He also asks whether pneumonia may not follow influenza as the result of the inability on the part of the lungs to rid themselves of some of the toxic material through depression of the cough reflex. Also he suggests that post operative pneumonias may possibly be caused by the temporary abolition of the cough when a general anesthetic like morphin is employed.

It is questionable whether the trachea and smaller bronchioles may participate in a cough reflex. Nothnagel mentions that irritation of the laryngeal mucosa above the vocal cords produces spasmodic contraction or closure of the glottis but no cough. But coughing does occur through irritation below the cords. The most sensitive area is the interarytenoid mucous membrane and the bifurcation of the trachea (the carina). Sensitiveness to the cough reflex decreases from the larynx down to the pulmonary parenchyma. There is a teleological reason for this since the further down the trachea the foreign substance resides the less effective is the action of the cough. Mackenzie agrees that only when the mucous reaches the upper trachea or larynx does the expulsion take place. He cites the absence of cough in pneumonia as evidence of this. Many will disagree with this statement and other explanations must occur to every one. Amongst bronchoscopists the opinion is held that at the carina or ridge at the bifurcation of the trachea resides a spot extremely sensitive to irritation and acting as a receptor surface for the cough reflex. Some years ago in my laboratory Imperatori carried on some work with the idea of attempting to explain the cause of sudden death in certain cases where bronchoscopic examination had been made and where no lesion or puncture of the lungs was in evidence. The results of this investigation were presented to this Society in 1916 as a candidate's thesis. Imperatori showed that stimulation of the carina caused increased rate and amplitude of respiration. From other findings the conclusions were tentatively advanced that sudden pulmonary collapse might reflexly inhibit not only respiration but the heart as well. The question still remains unanswered and an enticing field of work still awaits an enthusiastic laryngologist.

Laryngeal Spasms of Reflex Origin.

That spasm of the adductor muscles does occur as the result of reflex stimulation, seems to be well established. In the young

individual it may occur from a great variety of causes all acting to elicit the reflex, but in the main the neurotic factor is to be emphasized. In pertussis the adductor contraction is said to be the result of central stimulation; but in most of the other conditions the sensory area from which the reflex originates is the larynx, nose or pharynx. It appears to be true that local disturbances in other parts of the body when occuring in connection with a neurotic condition may also act as causal excitants. In adults the so-called nervous largyngeal cough is to be explained in a similar way. It is interesting to note that this reflex generally ceases at night or when the patient's attention is diverted. This introduces a matter of some importance concerning the mechanism of the reflex involved in the cough. Jendrassik divides all reflexes into the spinal type which includes the tendon, periosteal and joint reflexes, the cerebral types such as the cutaneous reflexes and the complex type of which sneezing, coughing and vomiting are examples. One characteristic of the latter two types as distinguished from the tendon reflex is the necessity of the sensation of tickling in connection with eliciting the reflex. Apparently sensation, or a so-called cerebral component is necessary to the completion of the reflex. This raises the question as to whether the cough may be aroused without consciousness. Sahli, in his diagnostic methods, states definitely that "completely unconscious individuals never cough or sneeze." This raises the further question as to what is meant by complete unconsciousness. Some observers speak of the degree of unconsciousness or anesthsia necessary to inhibit the cough. It is to be assumed that in the last stage of anesthesia, reflex coughing could not occur since the muscles are thrown out of action; but in the opinion of anesthetists the individual may cough in light surgical anesthesia. Imperatori, at my suggestion, recently examined eighteen cases in anesthesia with the view of determining the relation of the disappearance of the corneal reflex to the ability of eliciting the cough. He informs me that coughing may be reflexly produced after the cornea has lost its reflex. In this he agrees with Wright and Smith who also state that the posterior wall of the larynx is most susceptible to irritation. Whether the pharynx or larynx first ceases to serve as a sensifacient area for the cough and how deep must be the anesthesia in order that the reflex drops out, Imperatori is unable to decide at present. If all the above is true the statement of Sahli must be incorrect. It had always been my

assumption that coughing during supposed sleep was the result of the irritation which brought the individual into a state of semi-consciousness during which the sensation of tickling actually came into consciousness and the cough resulted. The absence of the nervous laryngeal cough in individuals when their attention is diverted seems to add some evidence to the view that sensation is a component in production of the cough, though I must admit that the subject is still an open one.

Another condition of laryngeal origin is the vertigo which sometimes accompanies the abductor spasm of the glottis. Among other indefinite etiological factors are mentioned tobacco and alcohol. Apparently the vertigo is merely a symptom induced by impaired cerebral circulation resulting from interference with blood flow in the lungs from the cough.

In certain cases of acute laryngeal edema the disturbance may also be considered in the same light as the production of a mucous edema of the nasal mucous membrane, either as the result of a pure reflex involving central synaptic connections in the medulla or through an axon reflex as explained before.

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DISCUSSION.

Dr. Greenfield Sluder, St. Louis, Mo., said he was greatly interested in this paper. The only portion he could comment upon was relative to the syndrome of the spheno-palatine ganglion. Dr. Jackson had seemed to assume for it attributes of the sympathetic. It is a difficult question to decide. It is opposed to the views of Anderson and other workers. They regard the sympathetic as an efferent system, purely. If the cut ends are stimulated, they produce no response. The speaker asked if Dr. Jackson assumed afferent fibers for the sympathetic.

Dr. Jackson replied that he did not hold that the autonomic is purely efferent, but thought that there must be afferent fibers from the visceral organs. He called them visceral, but one finds there are statements that there are afferent autonomics.

Dr. Sluder said he had used the term sympathetics carelessly; it was the autonomic that he had in mind. If one could assume this to be a fact as Dr. Jackson said, that there are afferent paths for the autonomics that explains the painful syndrome through the nasal ganglion and the asthmatic reflexes from the nose. Stimulation of the trigeminus would produce influence on the pulmonary viscera. One might anesthetize the trigeminus in the nose, but the asthma is not influenced by the procedure. That asthma may be controlled by anesthesia of the nasal ganglion has been proven. Dr. Sluder asked the nature of the multipolar cells in the nasal ganglion. The post ganglionic neurons are connected with the nasal ganglion, and the pre-ganglionic cells, as explained by Dr. Jackson are from the geniculate ganglion.

Dr. Jackson said that the preganglionic cells from the 7th originate in the pons, but the preganglionic neurons have nothing to do with the autonomic supply. The geniculate is a sensory ganglion and has nothing to do with the autonomics. The fibers just pass through that location.

Dr. Sluder said he understood that the geniculate is a sensory ganglion. The explanation of the phenomena by sensory overlapping was not mentioned. Dr. Cushing made a mistake last year when he assumed that a headache which the speaker assumed to have a sensory significance, was a spilling over from the trigeminus. The question arises, can one have the trigeminus as a provoking factor of pain in the neck. Patients complain of a feeling as of a ball of fire between the scapulae, with no pain in the trigeminal distribution.

Dr. B. N. Colver, Battle Creek, Mich., said he had seen cases where merely touching the anterior end of the inferior turbinate caused pain at the end of the eye brow on the right side. A connection had been established between two spots; one in the inferior turbinate on the border, which resulted in a spasmodic cough in the larynx that began as a tickling as of a feather. Touching this spot would produce spontaneous irritation, which was stopped by anesthetization. The speaker began to study these cases of non productive irritative cough in the larynx which was found due to a sensitive spot in the posterior wall of the pharynx, level with the floor of the nose. By treatment with an applicator the cough could be relieved. In one case of pertussis in a boy of 8, in whom the frequent paroxzysms caused complete non-retention of food on the stomach, the impulse was controlled by treatment of a sensitive area in the pharyngeal wall. After the catarrhal stage has passed the spot can be reached and treated. In the early stage of pertussis this is impossible.

Dr. L. K. Guggenheim, St. Louis, Mo., said that until recently he had not been able to prove relief of pain by injection of the spheno-palatine ganglion, but lately he had had a dramatic case in an old lady of 70, who was siezed with violent pain in the ear, tenderness over the mastoid especially over the emissary vein. Examination of the ear was difficult on account of the excessive pain, but no pathology could be made out. The pain had followed an attack of influenza. He ordered codein, without relief, and finding nothing to account for the pain, thought of the sphenopalatine ganglion. He injected it with a five percent cocain solution, with complete immediate relief of pain, without any further recurrence.

SOME POINTS IN THE COMPARATIVE ANATOMY OF THE NOSE AND THE ACCESSORY SINUSES WHICH ACCOUNT FOR THE VARIATIONS IN THESE STRUCTURES IN MAN.

(Lantern Slides)

By J. M. INGERSOLL, M. D., Cleveland, Ohio.

In its simplest form the olfactory organ consists of two depressions, in the integument of the snout, which open externally, but do not communicate with the mouth.

Fish. The olfactory organs of the fish are two rather regular oval cavities separated from each other by a thick median wall or septum. Each of these cavities opens externally, and in some fish this external opening, or nostril, is divided and thus an anterior and posterior nostril is formed. There is, however, no communication between the nasal fossae and the mouth. The septum supports a number of radiating processes extending into each nasal fossa. These processes are covered with olfactory mucous membrane in which the olfactory nerves end and they correspond with the olfactory turbinals of higher vertebrates.

Dipnoi are fish which resemble amphibia in some respects. They live in shallow streams and bury themselves in the mud during the dry season. The nasal fossae open into the mouth, and a very primitive lung is developed which functionates while the fish lives in the mud and no longer receives its oxygen thru the gills. In this way anterior or external and posterior or internal nostrils are differentiated and a free passage is formed through which air passes into the lungs and the olfactory organ becomes a part of the respiratory system.

Reptiles—The Turtle. The character of the nasal organ is modified in reptiles, by the change in the method of respiration. The nasal fossae open into the mouth and are differentiated into an olfactory and respiratory portion.

The olfactory organ of the turtle consists of two symmetrical cavities with a relatively thick partition in the median line.

Each cavity is subdivided into a respiratory and an olfactory region. The respiratory part is a tube like structure which extends downward and backward and opens into the anterior part

of the mouth, thus forming the posterior nares. Around these two openings into the mouth there are numerous finger like projections which, when they are brought into apposition, close off the posterior nares from the mouth.

The olfactory part of each fossa is a round cavity, above the respiratory region, and partially separated from it by a prominence projecting out from the lateral wall. This prominence corresponds to the turbinal in other vertebrates.

In more highly developed reptiles the secondary palate is formed by the union of the palatine processes of the superior maxillary bones with the pterygo-palatine arch. Thus a secondary roof of the mouth is formed distinct from the sphenoidal or primary roof. The cavity thus formed below the sphenoidal bone, prolongs the nasal chambers backward so that they open into the nasopharynx, instead of the mouth.

Birds—The Duck. In most birds the anterior portions of the nasal fossae communicate with each other, the septum being incomplete and posteriorly the two fossae have a common opening into the pharynx, which is surrounded by finger like projections similar to those around the posterior nares of the turtle.

In the duck there are three turbinals in each fossae. The anterior one is an inverted "T," attached to the roof of the nasal chamber and is suggestive of the maxillary turbinal with a double coil seen in the higher forms of vertebrates. It is, however, covered with stratified epithelium, and is called a pseudo-turbinal.

There are two ethmoidal turbinals. The anterior inferior one has a double coil and occupies the greater portion of the olfactory part of the nose. The superior ethmoidal turbinal is a simple prominence with two irregular projections extending downward.

Mammals. The turbinal structures in mammals exhibit great variations. In microsmatic mammals (animals with an acute sense of smell) the turbinals attain their highest degree of development and are very complicated structures.

Panther. The turbinals in the American panther are a good example of this high degree of development. The nasal fossae are separated from each other by a thin, straight septum which unites with the cribriform plate posteriorly. Inferiorly the septum is formed by the vomer and presphenoid, and superiorly by the descending plates of the frontal and nasal bones. Along the articulation of the perpendicular plate of the ethmoid with these descending plates, a lateral process extends out at right

angles into each fossa and supports a considerable portion of the olfactory turbinals and partially subdivides the olfactory portion of the nose into two superimposed cavities.

The maxillary turbinal is in the anterior part of the nasal fossa. It springs from the superior maxillary bone by a slender pedicle, which extends out into the fossa, nearly parallel to the floor of the nose, and divides into two branches. The superior branch curves upward, making one complete coil. The inferior branch curves downward and makes two turns within itself. The whole structure is a typical example of the double coiled turbinal, which is present in many mammals. It is the homolog of the inferior turbinal in man.

The naso-turbinal is a rather simple structure, which projects downward from the nasal bone and overlaps the maxillary turbinal and the anterior part of the ethmoidal turbinals. Its homolog in man is the agger nasi, a small bony ridge anterior to the middle turbinal.

The ethmoidal or olfactory turbinals are very complex structures. There are six of these turbinals in each fossa. They radiate from the convex surface of the cribriform plate, and the extent of their surface is tremendously increased by almost innumerable folds and branches, which make an intricate labyrinth occupying the posterior part of each nasal fossa and constitute the olfactory portion of the nose. Branches from these turbinals extend into all of the accessory sinuses.

The posterior portion of each nasal fossa is subdivided into two superimposed cavities by the lateral wings of the vomer. The superior cavity is the olfactory portion of the nose, and contains most of the olfactory turbinals. The inferior cavity connects the respiratory portion of the nose with the nasopharynx.

Dog. The maxillary turbinal in the dog is attached to the superior maxillary bone by a single shelf like process extending along the external lateral wall of the anterior portion of the nasal fossa. Numerous curved branches are given off which nearly fill the anterior nares and present a very large surface of respiratory mucous membrane. The double scroll shape of the maxillary turbinal is supplanted by the numerous branches, except in the posterior inferior portion of the turbinal. Here the scroll shape still persists.

The ethmoidal turbinals are very complex structures consisting of many thin delicate scrolls and branches of which at least

five radiate from the convex surface of the cribriform plate. Some of the numerous branches of the ethmoidal turbinals extend into all of the accessory cavities of the nose. One scroll is supported by a process which is attached to the external lateral wall of the maxillary sinus.

The maxillary sinus is hollowed out of the superior maxillary bone and it communicates with the nasal fossa through a large opening which extends from the floor of the cavity to its roof. The process which springs from the lateral wall of the sinus and supports a part of the ethmoidal turbinal extends into the nasal fossa through this wide opening. The frontal sinus is situated between the two plates of the frontal bone and its anterior inferior part is occupied by a double coil of the ethmoidal turbinal. The two sphenoidal sinuses are separated from each other by a rather thick septum and are nearly filled by scrolls of the ethmoidal turbinal.

The posterior part of the vomer is expanded into two lateral wings which unite with the ethmoidal bone and divide the posterior part of each nasal fossa into two superimposed cavities. The superior cavity contains most of the ethmoidal turbinals and is distinctly an olfactory chamber. The inferior cavity is a tube like structure forming a direct communication between the anterior half of the nasal fossa and the nasopharynx.

The function of the accessory cavities of the nose in the panther and the dog is to provide space for the tremendous development of the olfactory turbinals, to conduct air over the external lateral surfaces of these structures and to furnish a system of drainage for them.

Apes. The ape's nose is a very much degenerated structure. The maxillary turbinal is quite similar in outline and position to the corresponding turbinal in man. In some apes it has only a single inferior coil and in others it still has a small superior coil also.

The ethmoidal turbinals vary considerably. There may be only one, or two of these present and in a few apes a rudiment of the nasal turbinal shows quite distinctly. The numerous fine convoluted branches of the ethmoidal turbinals which are present in lower animals and the fourth and fifth ethmoidal turbinals have all disappeared.

The maxillary sinus is usually fairly large and opens into the middle meatus through a small ostium. The walls of the sinus are smooth and the sinus contains no ethmoidal turbinal tissue.

Usually in apes there are no frontal sinuses, and the frontal bone is cancellous. The chimpanzee, however, has two fairly well developed frontal sinuses. The ethmoidal cells vary in size and number. In some apes there are none and the ethmoidal turbinals are very rudimentary. In others there are several ethmoidal cells and the ethmoidal turbinals are more highly developed.

The sphenoidal cells vary also and are present in those apes which have ethmoidal cells, and in the orang-outang the sphenoidal cells show rudiments of ethmoidal turbinates.

Man. The general characteristics of the accessory cavities of the nose in man are too well known to need a description here. They show numerous variations many of which are reversions to more primitive types. The absence of one or both frontal sinuses correspond to the absence of these sinuses in most apes.

The ethmoidal cells are formed by the union of the numerous branches of the ethmoidal and adjacent bones and occasionally show small irregularities which suggest rudiments of the ethmoidal turbinal scrolls which are well developed in some of the lower animals.

The subdivision of one or both sphenoidal sinuses correspond to a similar division of these sinuses in some of the lower animals in which the ethmoidal turbinals extend into the sphenoidal sinuses and thus produce numerous subdivisions. The irregular ridges sometimes present on the lateral wall of the maxillary sinuses are probably rudiments of the ethmoidal turbinals which in some animals extend directly into these sinuses and are supported by attachment to the external lateral walls of the sinuses.

The accessory sinuses in the lower animals develop in connection with the olfactory portion of the nose and form channels which conduct the inspired air over the external lateral surfaces of the olfactory turbinals and in this way increase the acuteness of the sense of smell. In the higher orders of mammals, particularly the apes and man, the olfactory turbinals have degenerated and receded from the accessory cavities and the cavities are left with comparatively smooth walls and their functional activity is lost.

In man the accessory cavities are almost completely shut off from the nose. Their functional activity ceases and they remain as rudimentary structures.

DISCUSSION.

Dr. H. P. Mosher, Boston, Mass., said he had had the pleasure of reading this paper before presentation. It was very systematically prepared, and deserving of study. There were one or two points new to him: one, that the outer wall of the maxillary turbinate had originated ethmoidal tissue. He thought it started at the inferior turbinate. An interesting thing is the presence of the turbinate in all the accessory sinuses. Dr. Ingersoll did not lay stress on the nasal turbinate. The difference in the turbinates of animals and men is the development of the snout. The turbinate running in under the nasal bone sometimes projects back between the ethmoid and the maxilla. A necessary factor is the structure of the inferior turbinate which is divided into coils in the herbivorous animals. It is surprising to find that in carnivorous animals there is also a coil. Another point is that the vomer when it goes back to the lateral portion is divided into an upper and lower chamber. The upper is the olfactory and the lower the respiratory portion. The dog has only a little pipe and when running can't get enough air without opening the mouth. Another point is that one has the ethmoid and sphenoid connected with the antrum. Dr. Ingersoll could have made the paper much more dramatic in regard to the olfactory sense in animals by increase of the air turbinate. In the sheep this is increased five times. In man it is increased one and one half times by the turbinates. The sinuses are rudimentary but they are of some use. They serve as resonance cavities.

Dr. Ingersoll (closing) said that the nasal turbinate in animals diverts inspired air into the pockets in the nose. One reason the dog opens its mouth when running, is that it has no sweat glands, and has to get rid of the moisture through its mouth. The shape of the turbinate is primitive. The dog has a branch which fills the anterior part of the nose, yet the curve still persists in the posterior turbinate.

FATALITIES FOLLOWING OPERATIONS UPON THE NOSE AND THROAT NOT DEPENDENT UPON ANESTHESIA. A STUDY OF THREE HUNDRED AND THIRTY-TWO HITHERTO UNREPORTED CASES.*

By HANAU W. LOEB, M. D., St. Louis, Mo.

The development of the operative field of laryngology has been one of the most startling advances of medical science. From being an occasional operator, the laryngologist has become an accomplished surgeon with an enormous operative material, far beyond that of any other surgical specialist.

The work has become so attractive, or, perhaps so remunerative, that there are today five thousand physicians in America, according to the American Medical Directory, who profess to be qualified to practice this specialty, one out of every thirty practitioners in this country.

In this large body all are not well trained, all are not skillful, all are not progressive, all are not mentally alert, all are not conscientious. Be this as it may, they have an important bearing on the well-being of the people of today.

Considering the tremendous operative activity of laryngology, it is important to gain some estimate of its mortality risk. The fatalities must be excessively small in percentage, considering the number of operations. The meagerness of the literature on the subject shows this; and yet the published reports are sufficient to justify a more searching inquiry with the view of assembling reports of fatalities which have not been published.

It has been deemed advisable to exclude in this paper the anesthesia fatalities which would bear approximately the same relation to the total number of cases as in other operations of equal grade, with the slightly added danger which surgical work about the air passages would entail.

Inasmuch as the writer has no desire in this paper to touch upon cases in which the diseased processes or the operative procedures were of themselves of a serious character, it was decided to limit the study to operations upon the nose and throat, as follows:

*By resolution of the Society, the writer has been asked to continue and extend these studies. Accordingly these cases will be included in those to be reported in the paper to be presented next year.

1. All intranasal operations on the walls of the nasal cavities and paranasal cavities, excluding pituitary operations and those performed for malignant disease, and

2. Tonsillectomy, adenoidectomy, incision for peritonsillar abscess. Endoscopic operations upon the larynx, trachea, bronchi and esophagus are excluded in view of the small proportion which the endoscopists bear to the total number of laryngologists. Furthermore, a study of such fatalities belongs more appropriately to an active endoscopist.

The writer became interested in the subject after the occurrence of a fatality in his practice following a submucous resection.

Report of Case.

H. C. C., an attorney, aged 52, consulted me on February 25, 1921, complaining of slight pain over the left ear for some months, which had become somewhat worse lately, and which was more severe at night. This right ear had been deaf for a year, while in the left the hearing had just begun to diminish. Examination showed: Hearing: R—Voice, 4 meters, whisper, 0.1; L—Whisper, 4. Weber lateralized to the right. Rinne—Negative, right; positive, left. Nasal septum was greatly deflected to the right superiorly and to the left inferiorly. Diagnosis of chronic catarrhal otitis media was made.

Inflation improved his hearing very much, and reduced the pain. It was felt wise to perform a submucous resection upon the septum in order to give him ample air for audition and for nasal respiration. Operation was accordingly performed on April 1, 1921.

He left the hospital feeling very comfortable on the next day. On April 4th he returned to the hospital on account of severe hemorrhage. The interne plugged his nose with gauze, which was allowed to remain until the following day when I removed it. In the afternoon he had a second severe hemorrhage, for which it was plugged again with gauze, by the interne. This was removed that evening without a recurrence of any hemorrhage except a very slight oozing. On the day after his return to the hospital he developed acute tonsillitis on both sides. The patient was feeling very comfortable until three days later, when he was taken with pain in the occiput, marked deafness and an abscess in his left ear.

On the following day he was delirious, absolutely deaf, with an abscess in right ear, but with no rigidity of neck. The head could then be rotated in all directions. No Kernig present. Examination showed bulging drum and hemorrhagic discharge from left ear. Paracentesis was performed, after which there was a copious purulent hemorrhagic discharge. During the evening manifest symptoms of meningitis appeared, and signs of facial paralysis with neck rigidity and definite Kernig. He died that evening.

Autopsy showed middle ear on both sides filled with thin purulent material; purulent meningitis marked over the petrous bone on both sides; tuberculosis, left lung; pneumothorax, left side; arteriosclerosis; infectious thrombosis of liver; chronic interstitial nephritis.

There was no lesion over the cribriform plate on either side, and the sinuses were free from pus.

Only one other case falling within the scope of this paper came to the writer's observation through his practice.

This was a man whom I saw in consultation with a laryngologist, who in attempting to remove the superior turbinate bone, had fractured the cribriform plate. Meningitis developed within twenty-four hours and death ensued on the following day.

Literature. As has been already stated, the literature of the subject is exceedingly meager, being made up mainly of reports of isolated cases, with occasional autopsy protocols and with an attempt to explain the occurrence of death in the case reported. There has been very little constructive work done looking towards frequency of a fatal issue and its reduction or elimination. The small number of cases reported militates against the expression of any conclusions which could be of much value.

The writer, therefore, undertook to assemble a larger number of cases, in the hope that a better understanding as to the frequency of such fatalities might be developed and that some conclusion of worth might be drawn. To this end a letter was sent to each of the five thousand practitioners of laryngology in this country, asking for reports of cases.

It is understood, of course, that such reports can not have the weight of those which come from a single controlled clinical source. Factors of error are bound to creep in, depending upon the multiplicity of observers, the designed or unintentional coloring of the reports and much unavoidable looseness of observation. And yet there has been a surprising helpfulness manifested, which expresses the profound interest in the subject and the earnestness of the reporters themselves. Four hundred sixty-seven answered that they had had no fatality whatsoever. Two hundred fifty made reports which this paper analyzes, and approximately 4,300 have thus far made no answer. As a result of this inquiry, I have reports of 332 fatalities, more or less complete and carefully collated to avoid duplication. When this large number is compared to the paucity of published cases, it is obvious from the very first that we have not given to the subject the importance or the seriousness it merits, nor have we had sufficient data to justify conclusions respecting it.

It is, of course, impossible to determine with positiveness the causes of death in all the reports assembled, because there has been no set formula to guide these and because of the variant conditions under which the observations have been made.

Altogether thirty-two autopsies are included, but these in many instances have been made by untrained men. The protocols are, therefore, not always of use. However, in spite of the possibility of inaccuracies as outlined, the writer feels certain that something of value can be had by the classification which has been made.

The following grouping of the fatalities is made with the number reported in each group:

I.	Meningitis	125
II.	Hemorrhage	55
III.	General Sepsis	20
IV.	Erysipelas	8
V.	Endocranium (excluding meningitis)	26
VI.	Respiratory Tract	43
VII.	Heart	5
VIII.	Miscellaneous	20
IX.	Undetermined	30
	Total	332

Meningitis.

Meningitis forms by far the most common cause of death. Although in the vast majority of the reports it followed nasal operations, in one instance it was the cause of death following adenoidectomy. The operation in this child, aged nine months, was performed to relieve convulsions, death occurring twenty-four hours later. It is likely either that the meningitis was present before the operation or that the endocranium was injured during the procedure.

There were four tonsil operations in this category. In considering tonsillectomy, no distinction is made as to whether or not there was a coincident adenoid operation. In two of these, an acute suppurative otitis media intervened between the operation and the meningitis, a possibility that can be readily understood. As is well known, a postoperative otitis media suppurativa is commonly of a virulent type.

In one of the other cases, the meningitis followed immediately after operation; in the other, ten days later, both rapidly succumbing to the disease. The causal relations in these two cannot so readily be explained.

Meningitis was the cause of death after 120 nasal operations as follows:

1.	Intranasal frontal operation	16
2.	Probing and irrigating frontal sinus	7
3.	Ethmoid operation, with or without resection of the middle turbinate	39
4.	Sphenoid operation	10
5.	Maxillary operation	1
6.	Resection of middle turbinates	15
7.	Submucous resection	13
8.	Removal of polypi	13
9.	Combined sinus operation	1
10.	Combined submucous and sinus operation	5
	Total	120

This is certainly an appalling list, which cannot be lightly cast aside. The preponderance of fatalities is altogether on the side of the operations which come into relation with the lamina cribrosa. Perhaps, the suggestion contained in the older books that the cribriform plate can be recognized by its hardness and resistance to the instruments may be somewhat responsible for the frequency of this fatal complication. In seventeen instances, the reporters noted the occurrence of fracture into the cranial cavity, but there were doubtless many instances where this accident was overlooked.

Several reporters saw the cerebrospinal fluid escape during the operation. Another undertook to remove the superior turbinate, without disturbing the middle turbinate. Another stated that he removed the entire cribriform plate which was sequestrated. Quite a few used the Ballenger ethmoid exenteration knife but discarded it after the fatality. In some, plugging the nose too long or too frequently appeared to be a factor. In others an acute otitis media suppurativa incident to the operation was the forerunner, as in my own case.

In most instances, meningeal symptoms appeared within twenty-four hours after the operation. Can this be explained on the theory of an infection without fracture into the cranial

cavity? If intracranial infection can make its presence known so soon after operation in the absence of fracture, what prevents its more frequent occurrence in the multitude of operations which disturb the bacterial relations? Are we wrong in asserting that the nasal cavities are most resistant to infections? Considerations such as these force us to the conclusion that most of the cases occurring within the first twenty-four hours are due either to a fracture or to the lighting up of a latent meningitis.

Of the nine reported autopsies made on patients dying from meningitis, fracture was found in five in whom the symptoms appeared during the first twenty-four hours; in one with no specification as to onset, and there was no fracture in the two cases sectioned in whom the symptoms appeared after forty-eight hours.

1. *Intranasal Frontal Sinus Operation.*

Considering the relative infrequency of the intranasal frontal sinus operation as compared with other intranasal operations, sixteen deaths from meningitis (13.3 per cent. of all the cases), are sufficient to show the dangerous possibilities of this operation and to warn those whose knowledge of the anatomy is not sufficient to justify their undertaking it.

These considerations may not apply to the removal and curettement of the ethmoid cells, but they are forceful in reference to the drainage of the frontal sinus in connection with any attempt to enlarge its canal or orifice. One operator reports two cases in which the operation was followed by meningitis within twenty-four hours, one dying two days later and the other three days later. Another reports a case almost the counterpart of the first of these. In another, the meningitis with unconsciousness supervened immediately after the operation with a temperature remaining above 105° until exitus three days later. In four, the symptoms appeared several days after the operation when the sinus condition had improved, justifying the opinion that there had been an infection without fracture. Another case is reported in which the operation was performed one week after the appearance of influenza, complicated by an acute frontal sinusitis. Two days later, meningitis developed and the patient died three days later.

2. *Probing and Irrigating the Frontal Sinus.*

This operation which many consider entirely free from danger is responsible for seven deaths, large in proportion to the number of such procedures undertaken. One of them developed the disease in a few hours, two in twenty-four hours, one dying a day later and the other two days later. The disease appeared two days after operation in two of the cases, several weeks afterward in another, and in one case the symptoms may have appeared before operation.

3. *Ethmoid Operation with or without Resection of the Middle Turbinate.*

In more than one-third of meningitis cases reported, this operation was specified as the cause. All types of operation are represented, and according to the reports, it is impossible to determine which type is most dangerous, with the exception of the Ballenger knife, which appeared to be most culpable either by reason of its mechanical construction or from inexpertness in its employment.

As no operations in which ethmoid curettement or exenteration was combined with those upon the septum or other sinuses are included in this group, the ethmoid procedure may be accountable for more fatalities than are here stated.

In nine the meningeal symptoms appeared within a few hours; in ten others within twenty-four hours; in five others within forty-eight hours; in eight of the remaining they occurred later, while in seven the time was unspecified. The onset of the symptoms has an important bearing on determining whether fracture into the cranial cavity has resulted, for, as has already been suggested, it is hard to conceive of a meningitis of lymphogenous or hematogenous origin occurring within twenty-four hours after the infection pathway has been first opened.

Escape of cerebrospinal fluid at the time of operation occurred several times. One of these was followed immediately by meningitis, another, after two weeks. Packing for eight hours to control post-operative hemorrhage is reported to have been followed in forty-eight hours by a fatal meningitis. One of the patients was a baby two years old. In one instance, the operator, a well known and most skillful laryngologist, after the onset of the meningitis, removed the cribriform plate in the hope of relieving the process. He found evidence of a chronic pachymeningitis.

4. *Sphenoid Operation.*

The sphenoid operation is looked upon by laryngologists as more hazardous than that upon any other sinus and they have imparted this view in large measure to their patients; hence, laymen are more prepared for a fatal issue in these than in any other type of intranasal sinus operation. A more a less extensive ethmoid exenteration practically always constitutes a part of the sphenoid operation, and naturally either may serve as an etiologic factor of the meningitis. The reports do not differ materially from those upon the ethmoid operation. In one case the meningitis came on before night and death occurred within seventy hours.

5. *Maxillary Operation.*

Only one intranasal maxillary operation was followed by meningitis, following what appeared to be a simple procedure in a long-standing process.

6. *Resection of Middle Turbinate.*

This has an unusually large toll—fifteen—for what is ordinarily considered a very simple affair. In only one was there a coincident operation performed, resection of the corresponding inferior turbinate. Three developed meningitis the first day, one died on the second day with symptoms of acute mania and one followed middle ear abscess and meningitis, supervening upon the operation. The last was performed by a general practitioner. The reports of this group are very incomplete, consisting almost entirely of a statement of the operation and cause of death. In one, cutting adhesions between the turbinate and septum resulted in meningitis in twenty-four hours and death thirty-six hours later.

7. *Submucous Resection of the Septum.*

Two factors combine to influence a meningitis following submucous resection: The perpendicular plate of the ethmoid and the exposure of the medial sides of the septal mucosa with exclusion of drainage brought about by holding the two exposed surfaces together. If there was much disposition to nasal infection in the nose, we should expect far more frequent expressions of that infection than we get from this operation. However, the fifteen cases here reported constitute a sufficiently large mortality rate from meningitis at least to be of much concern to

operators. In most of the cases reported, in which onset is specified, the meningeal symptoms did not appear until after the second day, substantiating the view that these cases are largely hematogenous or lymphogenous. In one, the symptoms developed the first day and the patient died on the next, the reporter being sure that a fracture was the cause. Autopsy revealed a puncture of the cribriform plate in another in which meningitis appeared soon after operation.

My own case, reported in this paper, comes under the category of secondary meningeal infection resulting from otitis media.

8. *Removal of Polypi.*

Although removal of polypi was one of the first nasal procedures to which our literature refers as a cause of meningitis and death, the thirteen cases here recorded will be considered a number far beyond the expectation of laryngologists. The natural inference would be that the forcible evulsion of the polypi with forceps must have caused a fracture into the cranial cavity or so close to it that infection was possible.

Several of the reports are exceedingly interesting. One patient became semi-stuporous within twenty-four hours. No autopsy was made but the operator thinks that a meningitis was developing at the time of the operation.

One surgeon removed a polyp from the superior meatus with a snare. He heard, what he calls, a rucking sound. Two days later the patient developed meningitis, which was soon followed by death. In another, an ethmoid exenteration had been done some time before; soon after the polypi were removed meningitis developed. The surgeon thinks that there was a necrosis of the cribriform plate which caused the acute meningitis by activation through the operative procedure. A patient immediately after the removal of several polypi from the nose fell into convulsions, and was taken with meningitis followed by death. In another from whom some polypi had been removed, autopsy showed polypi extending into the sphenoid. In another who died of meningitis eight days after operation, autopsy showed the lamina cribrosa on both sides as thin as paper through which the polypi in the ethmoid were visible. There was a very thin fracture running across the right cribriform plate and an abscess in the frontal lobe.

9. *Combined Sinus Operation.*

A combined sphenoid, maxillary and ethmoid operation resulted in meningitis and death in one instance.

10. *Combined Submucous and Sinus Operation.*

The operations performed were submucous and bilateral ethmoid exenteration, three, in one of which there was fracture of the cribriform plate with escape of cerebrospinal fluid; submucous resection, exenteration of ethmoid and intranasal operation on the frontal; and submucous resection with exenteration of ethmoid and intranasal maxillary operation.

Hemorrhage.

Hemorrhage constitutes the next most important group and is in evidence in both the nose and throat operations, as follows:

1.	Tonsillectomy (with or without coincident adenoidectomy)	43
2.	Adenoidectomy alone	2
3.	Incision of peritonsillar abscess	3
4.	Submucous resection	2
5.	Sinus operation	4
6.	Resection inferior turbinate	1
	Total	56

1. *Tonsillectomy.* It is certain that the forty-three deaths from hemorrhage after tonsillectomy do not by any means constitute all the unreported cases in this country. On the contrary, I am convinced that there are many more yet to be uncovered.

An analysis of the records shows that in sixteen there was a lack of post-operative control, that is to say, the operation was performed in the office and the patient sent home; or the parents insisted on taking the patient away; or there was lack of observation whereby the bleeding might have been recognized; or the operator left the city; or the physician in charge was unfamiliar with methods of producing tonsil hemostasis. Most of these patients would have been spared under other circumstances.

Hemophilia mainly unknown to the operator is charged with seven of the fatalities; in one the operator discovered too late that the patient's brother had died from hemorrhage after tonsillectomy, the basis of which was hemophilia.

In another instance, familial hemophilia was discovered after the death of the patient.

A fatal result ensued in four cases in spite of thoroughgoing ligation of the vessels. The fatality was immediate in three cases. Persistent uncontrollable bleeding occurred twelve times and fatal secondary hemorrhage four times, one being after three weeks. In one of these cases the bleeding occurred five days after the operation, coming from the nasopharynx, the patient never having been seen by the operator after the operation.

There were two instances in which there was an anomalous branch of the carotid. In one this branch was found to leave the main trunk of the carotid about the middle of the tonsil fossa, and the vessel was severed three inches from its origin. The section of the enucleated tonsil shows a portion of the arterial trunk of large caliber with two or three large branches, which supplied the upper portion of the capsule. Palpitation of the tonsil, according to the operator, would have discovered this situation.

One patient was an idiot; another had extremely high blood pressure.

From these reports it is obvious that much can be done to reduce the mortality from tonsil hemorrhage.

2. *Adenoidectomy.* Very little detail is given in these cases.

3. *Incision of Peritonsillar Abscess.* Of the three cases, one was in a child in which the incision was followed by persistent bleeding, which continued for some days and for which treatment was of no avail. Of the second no detail is given. The third proved to be a hematoma, which resulted from erosion of the carotid or internal jugular, death being immediate.

4. *Submucous Resection.* Two deaths from hemorrhage occurred after submucous resection; one was allowed to return home after three days, the physicians available not being expert enough to control it. As to the other, no details are given.

5. *Sinus Operations.* Hemorrhage resulted in death following puncture of the maxillary sinus, the bleeding being from both the gums and nose. A resection of the nasomaxillary wall came to a fatal issue by reason of hemorrhage; another death after removal of polypi, and still another after ethmoid exenteration. In none of these cases was there anything to anticipate a fatal hemorrhage.

General Sepsis.

The number of deaths from general sepsis is relatively small and is in keeping with the teaching that serious infections are rare in nose and throat operations, if we exclude meningitis. They include:

1.	Intranasal maxillary operation	2
2.	Submucous resection	1
3.	Tonsillectomy	16
4.	Incision peritonsillar abscess	1
	Total	20

1. *Intranasal Maxillary Operation.* In one of the cases, symptoms of general sepsis developed three days after operation for an acute maxillary sinusitis, which resulted in death on the following day from general peritonitis, the appendix not being involved. In the second case, the death occurred two days after operation.

2. *Submucous Resection.* In the single case reported, the fatal issue occurred eight days after operation. Packing had been retained for twenty-four hours.

3. *Tonsillectomy.* There is a growing feeling that sepsis is more likely to occur under local anesthesia than under general. This is in a measure confirmed by the reports which show that of those in which the anesthesia is specified, seven were local and two general. Localized swelling is characteristic of the majority, tracheotomy, though without avail, being required in two. In one, the pharyngeal tissues were found infected with colon bacillus; in another peritonsillar abscess developed; in another, chill, 106° temperature and abscess of the hip; another showed by cultures that there was an attenuated diphtheria infection.

4. *Incision Peritonsillar Abscess.* It cannot be determined whether the general sepsis reported was due to the operation or to the process itself.

Erysipelas.

The operator is accustomed to look upon erysipelas with more chagrin than any other complication unless it be preoperative. The grouping is as follows:

1.	Submucous resection	4
2.	Sinus operations	2
3.	Resection middle turbinate	2
	Total	8

1. *Submucous Resection.* In one of the submucous cases the middle turbinate was also resected. One showed symptoms one day after operation, the others much later.

2. *Sinus Operation.* Included in the sinus operations, were one of removal of nasal polypi by snare, in which erysipelas developed on the second day and one in which erysipelas was present at the time of the intranasal maxillary operation.

3. *Resection of Middle Turbinate.* One of the erysipelas fatalities following resection of the middle turbinate was in an old man with a chronic nephritis; the other patient bled considerably, requiring a gauze plug which was not removed until the following day.

ENDOCRANIUM, EXCLUDING MENINGITIS.

This includes the following:

1. Brain abscess	5
2. Cavernous sinus thrombosis	9
3. Cerebral embolus or hemorrhage	12
Total	26

In the absence of autopsy, the diagnosis of brain abscess and of cerebral embolus or hemorrhage must be more or less doubtful but in the grouping, the opinion of the operator is accepted.

1. *Brain Abscess.* In one the diagnosis was confirmed by autopsy, in another by operation. In two, the operation performed was removal of polypi. One of these developed a frontal lobe abscess by extension from subperiosteal and subdural abscess. The other, two days after the operation, became drowsy with slight elevation of temperature and pulse rate from 50 to 70. He died in four weeks. One case resulted from retention of gauze after an ethmoid operation. Another had ear abscess a week after operation from which the brain abscess resulted.

2. *Cavernous Sinus Thrombosis.* This extraordinary complication is probably due to a hematogenous infection which finds its counterpart in those cases of furunculosis and similar processes affecting the skin of the external nose vestibule and lip. Altogether there were nine cases, divided as follows:

Tonsillectomy	3
Sinus operations	2
Incision peritonsillar abscess	1
Resection inferior turbinate	2
Series of nose operations	1

Two of the tonsillectomies were followed very soon by the symptoms which developed very rapidly. The other was that of a young soldier who had had tonsillitis two weeks before. The operation was performed under local anesthesia without incident. On the fourth day he had a slight elevation of temperature and on the following day there was beginning exophthalmos with slight blurring of the disc on the left side. Symptoms rapidly increased until exitus on the seventh day after the operation. Diagnosis was confirmed by autopsy. The sinus operations were puncture of the antrum followed by orbital cellulitis and exenteration of the ethmoid.

There was nothing extraordinary in the incision of the peritonsillar abscess, except the unusual character of the complication. In one of the cases of resection of inferior turbinate, an adenoidectomy was done at the same time; in the other, the operation was bilateral. In the last case, three or four unspecified nasal operations had been performed at intervals of two days.

3. *Cerebral Embolus or Hemorrhage.* This complication is represented in the fatalities thirteen times:

Tonsillectomy	7
Incision peritonsillar abscess	3
Nose operations	3

Of the tonsillectomy cases, three were in patients over forty and appear to have been due to accompanying conditions. As to the other four, a reporter describes one as being some brain condition, probably thrombus; another is stated to have walked to the bathroom contrary to orders and succumbed after showing localized paralyses; another had hemiplegia from embolus of the right cerebral; the fourth died suddenly while sitting up in bed.

The three cases of incision of peritonsillar abscess are all reported by the same observer, who may have overlooked other possible causes.

The two nasal operations were upon patients fifty-six and fifty-eight years of age. One, upon whom a submucous resection was made, had been a hard drinker with a blood pressure of 230; the other died suddenly when he was put back to bed after an ethmoid exenteration.

Respiratory Tract.

The fatalities coming under this heading are classified as follows:

1.	Foreign body	5
2.	Edema larynx and pharynx	6
3.	Pulmonary edema	1
4.	Pneumonia	17
5.	Pulmonary embolus and abscess	14
	Total	43

The operations concerned with fatal complications involving the respiratory tract are as follows:

Nose operations	10
Tonsillectomy	31
Adenectomy	1
Incision peritonsillar abscess	1
Total	43

1. *Foreign Body.* The five reports are as follows:

A submucous resection was performed under ether and the nose plugged with finger cot stuffed with gauze, which was inhaled and followed by death, the cause being unknown until autopsy showed the gauze stuffed finger cot in the primary bronchus.

A child died on the table after tonsillectomy from asphyxia due to clotted blood in the bronchi.

A very fat short-necked colored woman died after incision of peritonsillar abscess from pus entering the larynx and causing suffocation.

A tooth was dislodged during tonsillectomy under ether, subsequently causing death from inflammatory reaction.

A boy before tonsillectomy under chloroform had eaten a large amount of beans. Immediately after the operation he vomited about a pint of beans, some of which were inhaled and caused death from suffocation.

2. *Edema of the Larynx and Pharynx.* These cases may all have been septic though some were exceedingly rapid. In one the edema was so rapid and severe that the patient died within twelve hours after the operation; another for whom tracheotomy was done within twelve hours survived two days. One died two

weeks after operation, and two others in one week; still another died of edema glottidis one month after operation.

3. *Pulmonary Edema.* This was found associated with a miliary tuberculosis on autopsy after a death from tonsillectomy.

4. *Pneumonia.* When pneumonia follows an ether operation, it is usually assumed that the anesthetic is responsible. In practically all of the cases here reported the anesthetic etiology may be discarded. One was due to the inspiration of a piece of cartilage in a submucous resection under ether causing foreign body pneumonia; two others were septic pneumonia from tonsillectomy, one under gas-oxygen and one local; another after an intranasal antrum and frontal operation under local anesthesia developed septic pneumonia. The complication followed a curettement of the ethmoid and also an intranasal maxillary operation. Among the conditions blamed for pneumonia after tonsillectomy may be mentioned aspiration, influenza, recurring bronchopneumonia, a coincident peritonsillar abscess. All of these cases but one were under local anesthesia.

5. *Pulmonary Embolus and Abscess.* Pulmonary abscess has been held as due to the general anesthesia, but the investigations of Hunt and Cutler show that it usually results from embolus. Hence it belongs under the category of this paper and not under that of anesthesia. Many more cases of pulmonary abscess will undoubtedly be discovered if the investigation is extended. All the cases of pulmonary abscess following tonsillectomy were general except one which was local and one not specified. In the one reported exception the complication followed a resection of the inferior turbinate under general anesthesia. One reporter had three cases. Another gives the record of autopsy which showed suppuration of the posterior ethmoid and sphenoid present. Of the four cases of pulmonary embolism, two were confirmed by autopsy.

Heart.

The five cases of heart cause of death comprised the following: Angina pectoris, after tonsillectomy; dilation of the heart after removal of polypi; cardiac asthenia after tonsillectomy in a patient with grave heart lesions; cardiac infarct after incision of peritonsillar abscess and rupture of heart confirmed by autopsy in a submucous resection.

MISCELLANEOUS.

This list includes the following:

1.	Acidosis	1
2.	Delirium tremens	1
3.	Dilatation of the stomach	1
4.	Diphtheria	1
5.	Epilepsy	1
6.	Hyperthyroidism	2
7.	Orbit injury	1
8.	Poisoning	1
9.	Scarlet fever	3
10.	Shock	1
11.	Status lymphaticus	5
12.	Uremic coma	1
13.	Uterine hemorrhage	1
	Total	20

The acidosis fatality occurred in a diabetic upon whom a local tonsillectomy was performed.

The delirium tremens followed a sphenoid operation.

Acute dilatation of the stomach resulted, according to the reporter, from a heavy meal a half hour before an ethmoid exenteration.

Diphtheria caused the fatality after a tonsillectomy in a man fifty-five years of age.

The operator had no knowledge of previous occurrence of epileptic seizures in a patient who died in an attack after a resection of both turbinates.

Hyperthyroidism unrecognized previous to operation was held responsible for two deaths from tonsillectomy.

An inexpert operator pushed an instrument into the orbit in doing a submucous resection.

Another injected an overdose of adrenalin by mistake instead of novocain before a tonsillectomy, causing death.

Three cases developed scarlet fever after tonsillectomy, and died.

Shock was held responsible for a fatal issue after tonsillectomy in a patient with goitre.

Four tonsillectomies and one ethmoid exenteration succumbed to status lymphaticus, in which, of course, the anesthetic relation must be considered.

Uremic coma caused the death of a man suffering from an intense nephritis upon whom a submucous resection, nasomaxillary operation and an ethmoid exenteration had been done.

A woman with a high blood pressure expired suddenly from a profuse uterine hemorrhage sixteen hours after a tonsillectomy under general anesthesia.

Undetermined.

In thirty cases no cause was determinable, either by the physician in charge or by the writer from the reports submitted. The operative procedures concerned were the following:

1.	Tonsillectomy	16
2.	Adenectomy alone	1
3.	Exenteration ethmoid, with or without removal polypi	4
4.	Puncture of nasomaxillary wall, irrigation of the sinus	5
5.	Submucous resection	1
6.	Resection middle turbinate	2
7.	Combined tonsillectomy and submucous resection	1
	Total	30

The interesting cases in this group are those in which there was puncture of the nasomaxillary wall and irrigation of the sinus. They may well be compared with the sixteen already published and discussed at great length by Gording and Grove. There are doubtless some cases of status lymphaticus and unrecognized hemorrhage included in the group of undetermined causes.

Tabulation.

The table shows the cause of death in each type of operation. It is to be noted that tonsillectomy heads the list with 126, ethmoid exenteration being next with 46. The throat operations number 142; the nose operations, 190.

Reflections

Anyone who studies these reports, as the writer has, must be profoundly affected by them, particularly if he is at all sensitive to the unhappy experience which confronts the physician when one of his charges unexpectedly succumbs to an operation.

With this feeling I have set down what I term reflections, preferring to call them after this somewhat indecisive term rather than to hold them as well-grounded conclusions.

	Meningitis	Hemorrhage	General Sepsis	Erysipelas	Endocranium	Respiratory Tract	Heart	Miscellaneous	Undetermined	
Frontal	23									23
Ethmoid	35	1			4	1			2	46
Sphenoid	14							1		15
Maxillary	1	2	2	1	1	1			5	13
Resection Middle Turbinate	15			2		1		1	2	21
Resection Inferior Turbinate		1			2	1				4
Submucous Resection	13	2	1	2	1	3	1	1	1	25
Removal Polypi	13	1		1	2		1		2	20
Combined Sinus Operations	1					1				2
Combined Submucous and Sinus Operations	5				10			1		16
Combined Submucous and Resection of Either or Both Turbinates				2	1	2				5
Tonsillectomy	4	43	16			31	2	13	17	126
Adenectomy	1	2			1	1			1	6
Incision Peritonsillar Abscess		3	1		4	1	1			10
Total	125	55	20	8	26	43	5	20	30	332

They are presented in the hope that they may in some measure, if ever so slight, serve to reduce preventable fatalities after operations on the nose and throat.

I. *As to the Physician.*

1. Knowledge of the anatomy of the nose and throat cannot be learned by operating on the living patient—and yet, how many undertake to acquire it by surgery rather than by arduous study in the dissecting room.

2. To operate without this requisite knowledge is impossible for a man of conscience, not to mention the hazard to the patient.

II. *As to the Patient.*

1. The patient himself, and not the disease or the name of the disease should be studied.

2. Is he a proper subject for the operation? Is he too young or too old? How are his arteries, his heart, his lungs, his blood and his kidneys? Has he tuberculosis, syphilis, diabetes, carcinoma or some eventually fatal chronic process? Is the operation likely to benefit him, or it it worth while to operate upon him, considering his circumstances and environment, whatever such an operation may do for anyone else? These are potent questions of concern to the patient.

3. What might be termed the casuistry of operative indications is a good thing to study, for patients are not all alike; their response to operative intervention varies and the results good and bad cannot always be the same.

4. A good internist is a powerful help in the estimation of the patient's reaction. He has saved and will save many a life for the surgeon.

5. Under all circumstances where it is possible, the patient should be hospitalized. We can count many indeed, from the reports, who could have been saved if the operation had been performed in a properly equipped hospital.

III. *As to the Operation.*

1. Rigid attention to asepsis is essential, not by washing the field with antiseptic solutions, or by applying iodine, but by operating with an aseptic organization such as would be used in any other important surgical operation. The nose tolerates its own bacterial flora without much reaction but not extrinsic bacteria.

2. The sitting position is bad for nose operations; the patient's head is too easily moved; fainting is common; toxic symptoms from cocain occur readily; the physician's arms are in an awkward position, and instrument control is not always secure.

The semirecumbent position is far better. The patient need not be held; the range of head movement is greatly restricted, fainting and cocain intoxication are uncommon, the surgeon has better control over the operative field.

3. It is better to take out too little than too much—even if a second operation be required. There would have been less fractures of the cribriform plate if all of the operators had acceded to this position.

4. Multiple unrelated operations are an abomination. What is gained on the one hand is more than lost by the increased danger, the prolonged healing, the added complications. Furthermore when untoward symptoms appear, who can tell which operation is responsible for them?

IV. *As to the Postoperative Control.*

1. To operate and then to consign the patient to inexpert hands can have no justification. Many of the fatalities here recorded tell the tale of such practice, whether the surgeon leaves the patient, or the patient returns to his home without proper supervision.

2. The nurse or the mother or the attendant, whoever it may be, must be alert particularly in caring for those who cannot explain their symptoms.

3. A better surgeon is required to supervise the after treatment than to perform the operation.

V. *As to the Fatalities.*

1. Mankind has use for everything—even for adversity. Every fatality should call for an autopsy, carefully made and carefully recorded and then published, for the best way to avoid fatalities is to study those that occur.

2. The study and analysis of well kept histories and well planned autopsy protocols will bring about definite conclusions which will be of the utmost value in reducing the number of fatalities for which the less serious operations on the nose and throat are responsible.

DISCUSSION.

Dr. L. W. Dean, Iowa City, Ia., said he wished he had a better command of English to express his appreciation of Dr. Loeb's paper. Perhaps some of the orators of the Society would do this. Dr. Loeb had emphasized something very important, that one could not consider even a minor operation without danger. He had never told a patient that an operation was without danger of death. Status lymphaticus deaths are always associated with the anesthesia. In a thymic asthma case seen, the least manipulation of the nose would bring on an attack of asthma and breath holding, slowing of the pulse and cyanosis, turning up of the eyes. Then the patient recovered again. The speaker furnished an interesting and convincing case to Dr. Loeb. Twelve years ago an infant came into the clinical service for removal of adenoids, and the usual preoperative general examination was made. The child was prepared for operation and adenoids removed under chloroform anesthesia without accident. The child remained in hospital over night, and the mother stayed also. The baby nursed all right at one o'clock. At 3 o'clock it died. Autopsy showed embolism of the pulmonary artery. This thing might occur in even as mild an operation as incision of the finger.

Dr. Joseph C. Beck, Chicago, Ill., said if Dr. Loeb continued this work and made a text book instead of a paper it would be of the greatest value. In teaching methods, not sufficient emphasis is laid on preparation for operation. The A. M. A. course calls for two years training for specialization, but Dr. Beck said he did not find this sufficient with his assistants. He did not allow them to operate without his personal supervision. He had reported five deaths to Dr. Loeb, for one of which he was responsible. This was in submucous resection of the septum, with marked deformity. The man wanted the operation hurried and the septum operation was done first, followed by resection of the bones of the face and nose. This was too much and it was followed by meningitis and death. The preoperative examination, the speaker said, is important. Death is sometimes due to the general condition of the patient. Dr. Loeb's post-mortem work teaches a great deal. Doctors do not try hard enough to get the post mortems. Dr. Beck said that while he did not want to encourage the business of post-mortems, he would suggest that a prize be offered to the man who secured most autopsies, in spite of the family and the undertaker.

Dr. J. G. Dwyer, New York City, found this one of the most timely papers ever offered. He did not understand that complications as serious as mastoids were included. He said that as long as one operated on ethmoids and sphenoids in the dark, many deaths would ensue. The more he saw of ethmoids, the more respect he had for them. In seven cases of meningitis, he thought it was due to injury of the cribriform plate. The organisms present in the nose are limited by granulation tissue, but with injury, one throws a virulent organism into a fruitful field and gets meningitis in 48 hours. The tonsil cases (excluding hemorrhage) are of interest. The tendency in New York is to remove tonsils in the presence of acute cervical adenitis. This is criminal, as the swelling is due to nature's efforts to localize infection. One case with Ludwig's angina died of general sepsis. In a peritonsillar abscess case a thrombosis of the jugular vein occurred. One danger of opening retropharyngeal abscess is from inhalation of pus into the lung. The speaker agreed with Dr. Dean that there is no operation without danger.

Dr. J. A. Stucky, Lexington, Ky., said nothing more important than this paper had been presented. He thought a tinge of guilt ran through all their minds. Some of them had been operators before they were efficient doctors. Now they had waked up to the fact that there are

no minor operations. No more dangerous, treacherous field exists than the attic of the nose, but it will take care of itself if given a chance. A drop of yellow fluid does not mean the knife or curet. If one would give after treatment before operation there would be less sepsis. Three important points are: why to operate, when to operate, how to operate. The speaker said he could give Dr. Loeb many more cases. One point was that in every young adult coagulation time should be taken before tonsil operation. That had just been emphasized in a case where the assistant did not think it necessary. Dr. Stucky said he insisted upon it although the case was one of a healthy young professor. When the blood was taken, the coagulation time was 12½ minutes. Another point is the psychology of giving patients local anesthesia. Patients can be shocked to such an extent by improper psychological preparation, that the slight extra shock of the operation itself is too much and will tip the scale toward a fatal result. In this connection the speaker recommended the recumbent position.

Dr. F. R. Spencer, Boulder, Colo., said that he had to submit two cases to Dr. Loeb: one a case of meningitis, 48 hours after submucous resection and exenteration of the ethmoid cells by the Mosher technic. The post-mortem did not show fracture, but possibly there may have been a minute one. Pneumococci when confined to the nose are not malignant, but when occurring in meningitis are very virulent. Too many operators are satisfied with their knowledge of the ethmoid labyrinth, but this is a mistake. There are too many pitfalls.

Dr. Harris P. Mosher, Boston, Mass., said that he would express his gratitude to Dr. Loeb for the statistics, but wished he would change the title of the paper. The impression that these are minor cases, is unfortunate. The public have been trained to think sphenoid operations dangerous. Dr. Stucky, as usual, had taken the words out of the speaker's mouth. A mistake is made in allowing house officers to think they understand the ethmoid. Dr. Mosher said he was accustomed to address his post graduate class in some such terms as this: "Gentlemen, if I ever succeed in putting the fear of God into you, in regard to the ethmoid, I have not lived in vain." New England is supposed to monopolize the conscience of the nation. It would be well to break that monopoly and spread all over the country a more sensitive surgical conscience in regard to operations on the nose and throat.

Dr. J. F. Culp, Harrisburg, Pa., said he did not want to discourage Dr. Loeb, but he thought this report would be a drop in the bucket compared to what was to come. He would like to see a copy of this work put in the hands of every man in this Society, and of every man doing this work.

Dr. Coffin, New York City, said that the title should be changed. If he had known what was included, he would have given Dr. Loeb some cases.

Dr. Loeb (closing), said that if Drs. Mosher and Coffin would choose a suitable title, he would be pleased to adopt it. He expected a relatively small number of cases and selected the term "minor operations." If changing the title would help to educate the public he would be glad to consult with his advisors and make the change. The amount of interest shown was very pleasing and the help furnished was astonishing. In the letter sent out he had asked for cases in personal practice and in the vicinity. He got more replies as to the vicinity. He had promised to treat the whole matter in confidence and was pleased that the men had had confidence in him. At first he undertook to write to fifty men, his personal friends, but later thought the whole profession or rather specialty, should be addressed. The replies were so interesting, he had done nothing else than study them. He was glad to have Dr. Dwyer confirm his report upon meningitis.

AURAL EXOSTOSIS.*

By F. A. BURTON, M. D., Denver, Colo.

Although aural exostoses are known to have been fairly common centuries ago, the first effort to obtain comprehensive knowledge of the affection occurred in 1877, when J. P. Cassells claimed "to be the first observer who has given a rational explanation of aural exostosis, supported by clinical and pathological facts." In spite of some progress, its etiology is still largely speculative. The only etiological factors upon which the majority of investigators seem to agree are the influence of heredity and some forms of local chronic irritation, noninflammatory or inflammatory. As in otosclerosis, while the morbid anatomy is fairly well known; the pathogenesis is still in the realm of uncertainty. Therefore one ventures to give the imagination full play. It is interesting to note that the hypotheses advanced as to the etiology of otosclerosis are practically identical with those suggested in exostosis of the aural canal. Gray found exostoses not infrequently in the bony labyrinth, and reported cases of otosclerocis showing exostoses in the region of the promontory, oval and round windows. Katz and Fischer found exostoses on the ossicles in this disease. The writer ventures to think that a comparative study of the two conditions will impress one with the belief that the two processes differ fundamentally in the point of location only. Later on, this statement will seem less radical. There is abundant evidence that this disease was once very frequent among primitive races. In reviewing the literature and contrasting reports of prehistoric specimens with those of modern times, one can but be convinced that in the dim and misty past there were etiological factors which exist now in part only. To review the literature, present a case, report a comparatively large number of unreported exostoses in prehistoric specimens and to postulate what seems to the writer a logical theory as to the fundamental cause of the condition, is the purpose of this paper.

The review of the literature leaves one greatly confused by the lack of uniformity or system in the terminology. Each author has a different nomenclature, or none at all. Should

*Candidate's Thesis Recommended for publication by the Council.

not "canal" be used instead of "meatus"? Would it not be better for "exostosis" to take the place of "hyperostosis" and "osteoma", until a definite pathological basis for a classification is agreed upon by all? By some all bony growths of the canal are considered "exostoses", while others, the majority, use the more complicated terminology. Ballenger says that hyperostoses and exostoses are often spoken of synonymously although according to strict pathological interpretation they should be used to describe different lesions of bony tissue, the former being the diffuse thickening of the wall. Pritchard and Blake regard the hyperostoses as resulting more particularly from chronic inflammation, while Field speaks of the general thickening of the wall as the rarest form of exostosis. Sheppard reports two cases of "osteoma" and Ballance one "of the temporal bone blocking the outer part of the external meatus."

As in terminology, so in etiology, there is great diversity of opinion. All references credit these growths with being single, multiple, unilateral or bilateral—and in form broad based, flat or pedunculated, some even without a bony attachment (Shumacher, Jacquemant, and Delstanche). In frequency, they vary from five per thousand, to eleven per three thousand cases of aural disease—a comparative rarity today. Green believes they occur in fetal development, youth or early adult life. Dalby said they were congenital. Kerrison quotes Bezold, believing that the condition is rarely or never developed before puberty but in contrast, Hartman states that their growth terminates with the growth of the individual. As to the point of origin and location, Cassels believed that they always arise from the mastoid periosteum of the outer half of the canal; Pritchard and Blake, on the bony walls or the outer edge of the osseous meatus: Cassells and Pritchard again, and Ballenger, at the junction of the osseous and the cartilaginous canals. Kerrison, Blake, Jackson and Green have found them most often on the anterior or posterior walls—if single, on the anterior near the drum. Politzer alone mentions their existence on the floor. Ballance has published the only reference to one on the roof, i. e., Toynbee's case; and yet Cheattle limits them to the tympanic portion of the temporal bone. In point of location in the canal, Green places hyperostoses farther out than exostoses, and reports a case of exostosis in which the osseous canal was missing except the upper and lower posterior portions, the growth being attached to the tympanum or attic. He also

reported two other cases of bony growth located just within the meatus originating from the cartilaginous portion of the canal, and Lichtenberg one also.

Cassells held that these growths are due to a rupture of a subperiosteal abscess into the canal at the junction of the cartilaginous and bony portions, or through the cartilage, with ossification of the "granular growths" that spring up from the abscess opening. St. John Rossa in 1866, published four cases of "true exostosis", "morbid growths consequent on local irritation", in three being pus from the middle ear. Cocks reported a case showing bony metamorphosis in two polypi, in the course of an O. M. P. C. Pritchard held that one type of multiple exostosis originates in this manner but Weydner reported thirteen aural polypi examined microscopically and says that he has never been able to discover new bone formation in any that he has examined. Knapp definitely states that the pathogenesis is this—O. M. P. C., periostitis, polypi, and finally exostosis, citing a case of 34 years otorrhea. Ballenger, Politzer and Jackson believe that a fracture of the bony canal and the resulting periostitis, or a chronic middle ear suppuration exciting a secondary inflammation of the membranous canal may cause a fibrous or connective tissue thickening which after a long period of time might be metamorphosed into osseous tissue. Green believed them to be the result of a circumscribed periostitis, and found only in adults rather than youths. Carmalt regarded only the fibrous covered exostoses as inflammatory in origin, the cartilaginous being otherwise. McBride's case presented a history of repeated attacks of inflammation and Dalby's two and Lucae's one were associated with O. M. P. C., of long duration. One patient of Toynbee's suffered from a foul discharge for 20 years and he stated that in 1013 diseased ears examined, 14 showed narrowing of the external meatus as a result of bony tumors from the osseous walls.

In the opinion of Field, aural exostoses are caused by some local source of irritation, in many cases this being irritation by salt water repeatedly introduced through protracted bathing. With illustrative cases from districts near salt water, Dalby, Pritchard, Jackson, Connal, and Ballance add support to this theory which is opposed by Blake. He mentions the fact that the moundbuilders who suffered to a rather large degree from aural exostosis lived inland.

Ballanger, Pritchard, Blake, Field and Connal give syphilis, gout and rheumatism more or less standing as causative factors. But this relationship can only be held valid when other osseous tumors occur simultaneously in other parts of the body, the origin of which can be traced to the original disease (Politzer). By Kreible and Bergeim a metabolic study of a case of multiple exostosis was made with the result that the findings did not support the view of a relationship of this disorder to rickets or "to any primary disturbance of the general metabolism".

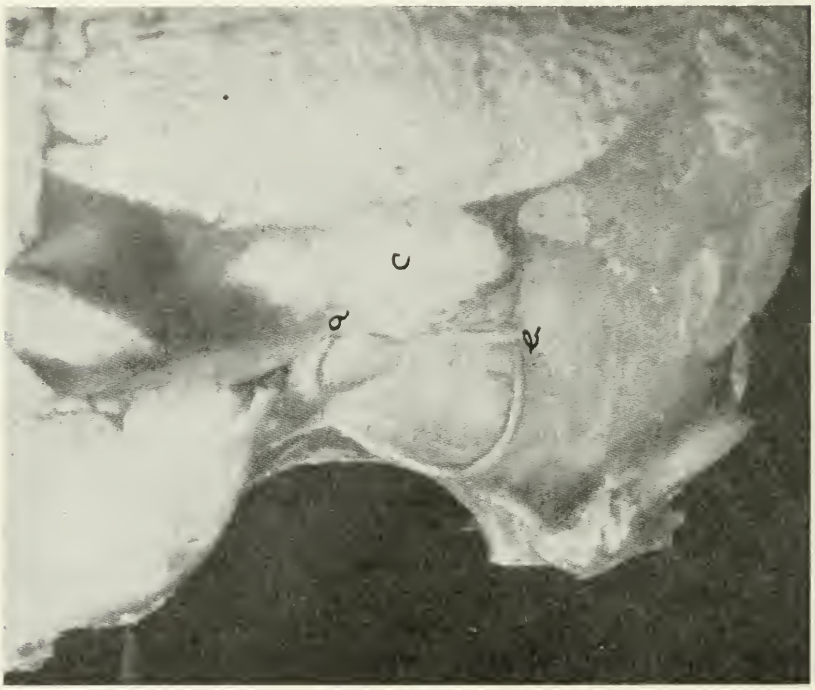
Several authorities have reported instances of a familial involvement, one case quite marked, over eight members of one group suffering from aural exostosis. Hartman exhibited two specimens of hyperostosis and held them to be anomalies of development for the reasons that (a) they appeared simultaneously in both ears, (b) that they were limited to the pars tympanica, (c) that there was a uniform consistency of the rest of the bone. Lake denies Hartman's theory but Politzer regards some cases as arising during the stage of development and ossification. Green, while mentioning a case of Jacks, reports three "true osteomata" of the mastoid, which he believes were probably of slow noninflammatory growth, possibly fetal in origin, originating in the tympanum or close to it. All were covered with normal skin. In two the osseous portion was overlaid with cartilage and in one, with fibrous tissue. In the discussion, Carmalt expressed his belief that the cartilage-covered ones were fetal in origin, and the other inflammatory. Whitney ascribed them to fetal cartilage rests. Peters had a case in which there was prominence of the supraorbital, temporal and suboccipital ridges, and hyperostoses of the nasal bones as well as of the lower jaw anterior to the masseters. In the discussion of this case, Jones suggested that general hyperostosis of the skull might be a stigma of degeneration. Politzer states that in many instances the cause is not ascertainable at all because the development is not accompanied by inflammatory phenomena, and we are seldom in a position to observe their growth clinically. In view of all this confusion as to the causation we may hide behind Virchow's statement that the etiology of these growths is an irritative disturbance of normal bone development. This "irritative disturbance" may be of any kind or variety, but the underlying question of why the condition arises in some and not in others with a similar aural disturbance can not be answered with certainty until the broad question as

to the etiology of all new growths, malignant or benign, has been answered. The balance in cell growth is in some way upset by a vaso-trophic element, or an irritative one, or by some factor which in many cases may be of such a nature that no history can be elicited. Naturally in a case that we say is idiopathic or spontaneous in origin, the patient can give no history except perhaps one pointing to a general bodily condition which may be present.

Historically interesting in treatment, is the use of postauricular counterirritation, leeches, dental burrs, snares, metal rods, and laminaria tents, the dilatation method being that of von Trölsch and Bonnafont. The complete removal of these growths surgically has become the recognized form of attack, marked deafness due to closure, marked deafness with disturbed hearing in the other ear, interference with drainage, and pain from pressure being indications for operation according to Politzer and others. Powell, however, suggests that one should not operate in cases of exostosis with a broad base because of the fear that the growth might extend into the tympanum. The chance of benefiting the patient should not be rejected because of this possibility, according to West. Selfridge in a personal communication referred to a case of his own in which it was necessary to do a bilateral modified radical operation after many minor operations had been tried. Finally one ear was converted into a radical mastoid on the advice of Duel and of Hammond, owing to the formation of an exostosis in contact with the annulus.

With the view of laying a further foundation for part of my conclusions, the development of the osseous canal has been studied. According to Kerrison, firm osseous union of the annulus with the rest of the temporal bone occurs during the first year of life, the free ends of the ring having united with the squama about the time of birth (Spalteholz). By deposition of new bone upon the outer surface of the tympanic ring, in the fibrous anlage of the bony canal the annulus is converted into a bony trough (Quain). Hertwig-Mark, in their textbook, call attention to the fact that the annulus gradually becomes broadened into a bony plate which fuses with the petrosal bone. This growth does not occur uniformly from the ring but mainly from two tubercles situated one near the upper end of its anterior limb, and the other at the lower posterior, so that the outer edge of the tympanic bone at this stage exhibits a notch

which by the subsequent union of the two tubercles is converted into a foramen. This aperture occasionally persists until adult life. While these changes are progressing the squamous part of the temporal bone situated above the membrana tympanica grows outward and comes to be nearly at a right angle with the vertical plate of the squamosa so as to form a roof. This process of formation is usually completed during the second year. Minot simply states that "the tympanic ring is incomplete on the lower anterior side and remains so for several years after

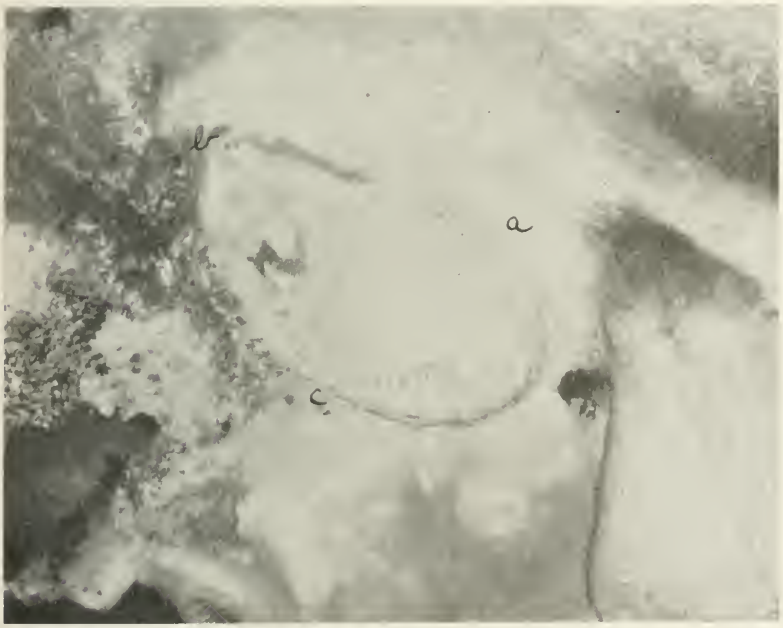


No. 1. Three months in utero. (x2) (left). The annulus is complete at this stage of development, open above. The anterior extremity is attached to the posterior end of the zygomatic portion (a), and the posterior end lies free just beneath the projection of the zygoma (b) backward along the base of the temporal bone (c). The anterior end is at least three times as wide as the rest of the ring.

birth." To illustrate the process of fetal development, the specimens in the San Diego Museum, Anthropological Department, have been photographed and the pictures with some descriptive matter are here presented.

Disregarding the many theories as to the direct cause, may not one assume that if the exciting agent be some form of

chronic irritation and the potentialities of all four walls be equal, the most dependent part, the floor, would be the wall receiving the maximum stimulation and hence should be the area most frequently productive of these overgrowths. But since the floor is almost never involved in the process, one seems justified in assuming other etiological factors than chronic irritation. It seems logical to believe that the exciting agent whatever its form, acts equally upon the entire canal but is responded to only by those points especially susceptible to the stimulus. In the



No. 2. Four months in utero. (x2) (right) The annulus (a-b) has doubled its width. The inferior portion lies against the petrous bone (c). Foramin magnum (d).

vast majority of cases reported only those areas responded to this stimulation, whatever it was, which during the development of the canal grew most rapidly. These places are the two ends of the annulus. May not these areas have retained the potentiality of more rapid development and response to any production stimulus? The answer "yes" is the writer's theory as to why the vast majority of aural exostoses are found along the suture lines between the canal and the rest of the temporal bone.

Case Report.

The case to be reported is as follows:

Mr. P., age 39. Scotch-Irish. Married. 5 children.

C. C. Almost totally deaf; mild tinnitus; sometimes very dizzy; paracucis Willisii.

F. H. Father died of "bleeding piles". At forty had a "growth, like gristle," removed from the right mastoid, because of rapid increase causing annoyance.

Brother died of "cancer".

No history of family deafness.

No. 3. Five months in utero. (x8) (right). Increase in growth of the ring very marked (a), with tubercle formation (d). The posterior end still is free. The squamous portion of the temporal bone is seen assuming a more horizontal position (c).

P. H. At 7 months gestation mother was injured, uterine hemorrhages of several days duration followed and emptying of the uterus was advised. Operation refused. Pregancy continued to term. At birth he "looked like he had no blood at all" and continued weak and anemic until about the age of five.

Injury to head at age of 2 resulting in unconsciousness of short duration, and spasms recurring every few days up to age of five.

Great swimmer since the age of 8, professional high diver from 14 until 1920.

"Sciatica" often. No positive veneral history. Measles, no ear involvement.

P. I. At sixteen patient noticed difficulty in clearing ears of water after diving. This gradually became worse until the sensation of an unremovable obstruction with terrific buzzing in the left ear nearly "drove him wild". The buzzing in the right ear began in 1915. The impairment of hearing in left ear began about the age of 19. His attempts to remove the water often caused bleeding and swelling of the canals. The canals have always been rather insensitive, with less than normal wax. Never any pain or earache. Hears high-pitched voices better than low. Canals previously lanced, diagnosis of "furunculosis".

Examination. Both canals closed externally by hard insensitive, immov-

No. 4. Six months in utero. (x7) (left). Very little change. Excessive growth at site of tubercle (a).

able growths covered by apparently normal integument. Hears only shout, the watch on contact, both ears.

A. D. C neg: C2 heard faintly: C4 heard, Galton O. K.

A. S. C neg: C2 neg: C3 heard: C4 heard, Galton O. K.

Weber to left, Rinne neg, both ears. Schwabach prolonged, both ears.

Slight transitory improvement on inflation.

Depletion of the canal walls so that a Yankauer Eustachian applicator with a bit of cotton on it could be passed, caused a marked improvement in the hearing. Nasal mucosa edematous; deviated septum in contact with the right middle turbinate body. Slight impairment of translucency of the right antrum.

Operation on the ears was refused but a submucous resection was agreed upon. Resection done in 1915, with relief of the nasal symptoms except some slight vasomotor disturbance at times.

Between this time and 1920, the patient came at intervals of a few months to have his canals treated. Finally in August, 1920, in the hope for relief from his increasing deafness and the unbearable tinnitus, he consented to an operation.

Physical Examination. Negative: no skeletal abnormalities noted.

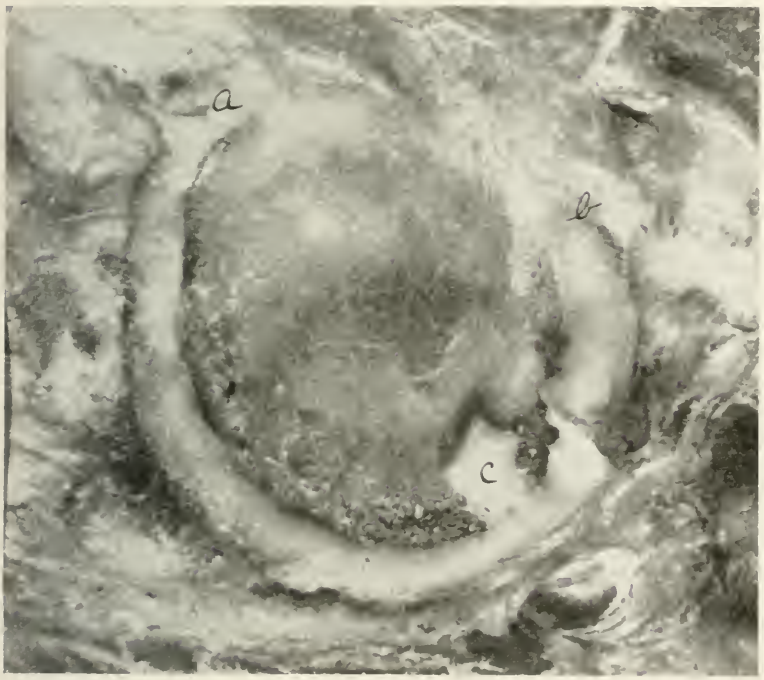
No. 5. Seven months in utero. (x7) (left). Annulus 1.75 mm. in width. The posterior portion in this specimen extends up and anteriorly along the face of the squamosa for several mm. (a).

X-Ray. "The auditory canal on the left is narrowed to about one-third of its normal size, probably throughout its entire length, but it is not occluded. The auditory canal on the right is almost completely occluded but this shadow is not dense and the occlusion is probably very thin. Certainly this occlusion does not extend throughout the entire canal." (Dr. L. C. Kinney).

Operation. A modified meatomastoidectomy on both sides, 8/17/'20. Wound healed in two weeks, leaving a fair sized canal on both sides. Hearing greatly improved subjectively.

	A. D.	A. S.
Voice	23 ft. (full length of room)	20 ft. (?)
Whisper	23 ft. (?)	20 ft. (?)
Acoumeter	23 ft.	9 ft.
C (64)	+	+
C 4	+	+
Rinne	+	+
Schwab.	Normal	Slight increase
Weber	To left	
Galton	Full range	Full range

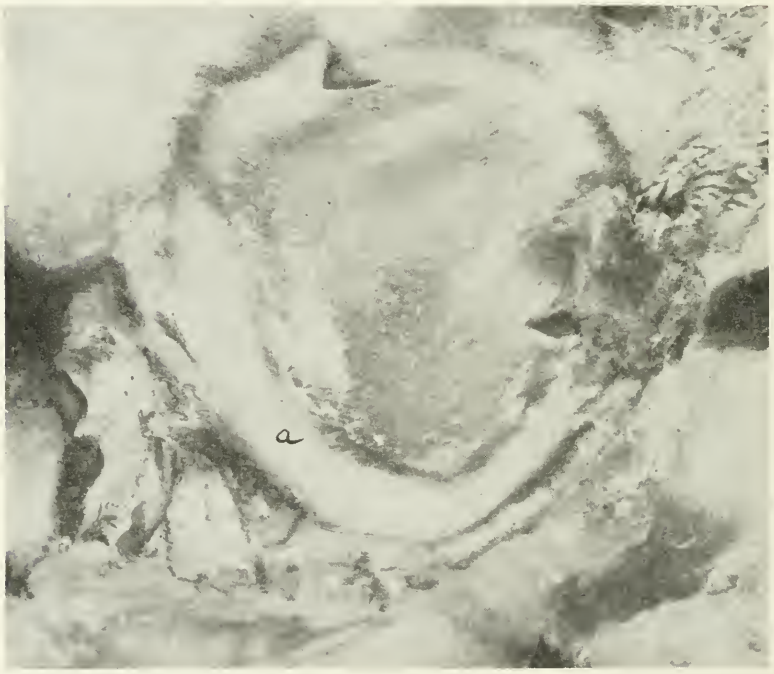
The patient returned July 13, 1921, complaining of a return of the deafness



No. 6. Eight months in utero. (x7) (left). Complete fusion of the ring with the temporal bone (a-b). Excess bone deposit in the drum extending in from the annulus. (This appears to be a similar condition to what was found in the case report.)

and tinnitus in the left ear. Right ear still normal. Fork tests showed a return of the left ear to about the original findings. Left drum hidden by two deeply placed hard insensitive noninflammatory growths, sessile, one on the anterior inferior wall and the other on the posterior wall. A narrow slit, only, existed between them. The patient was willing to have another attempt made to relieve the deafness and the tinnitus. A complete meatomastoidectomy revealed a dry cavity remaining from the previous operation. The entire posterior bony canal wall (i. e. what remained of it) was removed down to what was thought to be the annulus. A sessile bony growth 3x3x5 mm.

was removed intact. The small exostosis on the anterior inferior wall was not touched because of the feeling that enough had been done to relieve the symptoms. The wound healed promptly, the opening into the middle ear closing also. With the subsidence of all reaction the upper anterior portion of the drum was visible thru a triangular opening about three mm. on each side. At this time it became apparent that the exostosis removed with the posterior wall lay external to the annulus which as near as could be made out had become enlarged extending into and involving the drum. It had not extended inward so as to entirely occlude the tympanum for medication applied to the canal could be discharged thru the Eustachian tube into the throat (drum intact). At present the patient still complains of some tinnitus



No. 7. Nine months in utero. (x6) (left). The annulus is 2.25 mm. in width (a).

and very slight impairment of hearing and the functional examination shows:

	A. D.	A. S.
Voice	—	
Whisper	23 ft.	20 ft.
Acoumeter	23 ft.	20 ft.
C (64)		
C 4		
Weber	Not lateralized	
Schwabach	Normal	Normal
Galton	Full range	Full range

In the endeavor to discover the cause of this condition, especially since it seemed to be progressive on the left side at least, an investigation of the patient's general condition was made. Stereoscopic plates of the sella showed "apparently nothing which gives definite radiographic evidence of a departure from the normal in this particular case" (W. O. Weiskotten, M. D.) The Wassermann reaction negative. A complete study of the patient's metabolism was made by Dr. Robert Pollock whose findings were negative except for a slight degree of arteriosclerosis, very slight (negligible) creatinine retention and a like reduction of the alkali reserve. Facilities for a blood viscosity determination were not available. An examination of the patient's family gave nothing startling in the line of evidence towards a family tendency, yet bears reporting. One daughter, on the right showed what seemed to be

No. 8. Just after birth. (x7) (left). Marked tubercle at (a). Annulus is 4 mm. wide (b).

slight thickening of the posterior canal wall; a 2d daughter, the isthmus on the right narrowed antero-posteriorly; a 3d, thickening posteriorly and deeply. A son also showed some antero-posterior thickening on the right side. An older brother of the patient had normal canals. Wife's canals normal.

The pathological report of the specimen, by Drs. Grant and Wilson, is as follows: One portion showed "an osseous reticulum containing some slight amount of unchanged fibrous tissue. The osseous tissue itself contains a normal number of osteoblasts. In the few strands of fibrous tissue present, a very small number of fibroblasts may be noted. A second portion consists essentially of fibrous tissue with considerable numbers of fibroblasts present. There is a moderate amount of calcification present arranged in minute areas and in minute lamellae-like structures. No osteoblasts or osteo-

clasts were found. This condition seems to be a proliferative periostitis.' (Photomicrograph) This growth can be called an exostosis just as any bony growth from a long bone. The process may be different in that (as has been shown) the tympanic plate is a "membranous" bone and not cartilaginous. According to MacCallum, "the membranous bones are produced by a change in the function of certain cells which acquire the power of laying down an intercellular matrix, in which they bury themselves, and which in time becomes calcified." The microscopic examination shows the various stages of such a process from fibroblasts in fibrous tissue to osteoblasts in osseous tissue. This is to be expected since the process had been progressing up to the time

Photomicrograph of aural exostosis removed from patient.

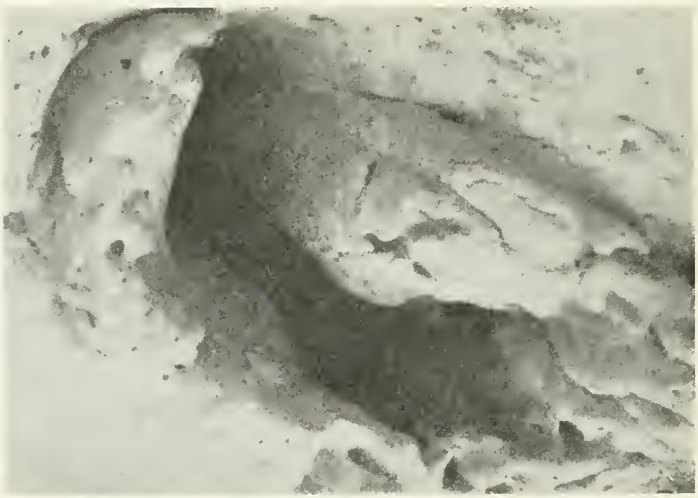
of the second operation. We have here, I should say, an idiopathic case with no evidence of local or constitutional irritation or basis of the new growth except the prolonged sea bathing.

Prehistorically considered, this subject is most fascinating. The literature naturally would be somewhat scanty, but I desire to make a resume of it before reporting the material studied. Blake in 1880 examined 195 skulls in the Peabody Museum of Archeology and Ethnology in Cambridge, from the stone graves of the mound-builders of the Cumberland Valley, Tennessee.

Thirty six showed exostoses, 12 in both canals, 9 in the right and 15 in the left. Of the 54 total growths, 42 were on the posterior wall and 12 rounded, principally on the anterior wall. They presented no evidence of syphilis. Ten of the cases came from one distinct locality, and 6 from another, pointing to a racial if not a familial tendency. He also investigated 108 California crania finding 5 possessing exostoses in one or both canals, and 5 cases out of 6 S. American crania, (Seligman), 2 out of 9 Marquesas Island skulls, and 1 Fox Indian (Welcher), 2 from Peru (Turner), and 6 in 334 Peruvian crania almost uniformly at the end of the canal and antero-superiorly or postero-inferiorly (Wyman). A continuation of his research, gave six exostoses in 37 crania all from one mound in Arkansas, again suggesting a family tendency. Here, too, there was no evidence of syphilis, the long bones being examined for that condition also. Jackson states that 25% of the skulls from the prehistoric mounds of North America show this condition, and found 25 cases in 450 North and South American crania, the majority being from locations near the sea. Virchow found 18 cases in 134 Peruvian skulls. In a personal communication, Dr. Bruno Oetteking of the Museum of the American Indian states that in their San Meguel and Channel Island collection the frequency is 23 per 1000 in the former and 13 per 1000 for the whole series. "The exostoses are pea-shaped, of solid texture and symmetrically situated at the anterior and posterior walls of the porus acousticus externus, thus corresponding to the endings of the originally incomplete annulus tympanicus. The porus is almost completely occulded; the pea-shaped exostoses are flattened at their point of meeting without, however, producing coalescence." With Dr. Oetteking's permission I give his conclusions derived from the Jesup material (No. Pacific) in the American Museum of Natural History. "Altho exostoses occur at an increasing rate in the different modes (of deformation of the skull) while the undeformed here have none, I have myself not come to a final conclusion as to a connection between deforming influences and the exostoses, since these happen also in undeformed skulls of other series. I should like, furthermore to call your attention to the fact that no exostoses have been found in the immature (juveniles and infantiles), which is according to R. Virchow (Crania ethnica americana) of some importance in judging the casuality of the phenomenon." The figures are as follows: Three types of deformations were examined. The lowest figures

were 2.3% among males only, or 23 per thousand. The highest was 32.8% among males and 15.4% among females of the same group, an average of 23.4% or 23 per thousand. In another type the females were twice as frequent in occurrence as the males with a final average of 1.9%.

The specimens to be reported are those of the Anthropological Department in the San Diego Museum, selected by Dr. Hrdlicka from the prehistoric cemeteries along the coast of Peru and mountains near the coast. In all, 4800 crania and parts of crania were examined in order to collect an exhibit of cranial pathology. Twenty-six deformed skulls showed 5 cases of exostoses in the



No. 425. Lomas, Peru. Three diffuse exostoses. As usual the tympanic plate is involved at the suture lines. (right).

canal and 1 in the middle ear just below the Eustachian orifice. All of them showed more or less antero-posterior narrowing of the canals, especially on the left. Fifty two crania chosen to show other lesions regardless of exostoses presented 5 occurrences. In one the external table of the mastoid had been destroyed by some pathological process uncovering a large cell containing a pea-shaped rough exostosis about 2 inches in diameter. Twenty two temporal bones and 2 skulls were collected specifically to show this condition. Only one specimen showed involvement on the floor, while four, in addition to growths on the tympanic plate had small scale or barnacle-like exostoses on the roof, a total of six separate excresences. The

total number of distinct exostoses was 84 and 46 temporal bones (including skulls) out of the 4800 crania examined. If we regard each temporal bone as coming from a different skull there was a total of 34 crania. Forty of the 78 growths limited to the tympanic plate extended to the annulus from the outer end of the canal, or over 50%. In many others the canal was so occluded that definite information could not be obtained in this

No. 439. Chicama, Peru. Two exostoses in the typical location along the suture lines. The anterior suture is open throughout ½ the entire length of the canal. (right).

regard. They were equally distributed between the anterior and posterior walls and all except 7 were situated at the sutures. tympano-mastoid, or tympano-squamous. The growths not at the sutures were never single but co-existent with those at the sutures. Four consisted of a diffuse thickening of the canal wall. The number of growths varied from one per cranium to 4 per temporal bone. The majority were roughly oblong and

extended the long way of the canal, taller at the outer end. The very small minority, in fact, only 5 were round or pea-shaped. Reduced to figures there were:

17.5 distinct growths per 1000 skulls.
9.5 temporal bones involved per 1000 skulls.
7. skulls involved per 1000 skulls.

The only suggestion as to relative prevalence among the males and females may be derived from the 52 miscellaneous crania. Four being males and one female. Considering the low percentage reported from the clinics and private practice of the present day, and postulating the premise that both in present life and in the selection of the exhibit an equal number of cases may not have been reported, we may conclude that this condition was more prevalent in prehistoric times. The literature has given figures in support of this conclusion. Present day statistics, it may be noted, refer to the percent of cases of aural disease while those of prehistoric man are those of crania in general irrespective of the aural condition.

Immediately anterior to the angle formed by the junction of the bony and cartilaginous portions of the external aural canal is the mandibular joint (Politzer), and it is a well-known fact that in mastication the movement of the lower jaw produces a change in the lumen of the canal, and that violence applied to the mandible at times causes fracture of the tympanic plate. The theories of Bruhl and Gray that stress may in some way be productive of the otosclerotic process, seem both ingenious and plausible. In a similar manner, the writer believes irritation to the aural canal due to mastication to be a contributary etiological factor of aural exostosis. Evidence of this may be gathered from the investigation of the life and habits of early man, who surely depended on nature's equipment. While he no doubt early became deft with his hands, it seems only natural to assume that the teeth became a third hand as it were bringing the jaws into strenuous use not only in masticating the tough uncooked foods, in cracking hard-shelled nuts, and assisting in preparing other necessities of life, but also as a weapon of defense. In most of the prehistoric crania examined, the greater involvement is at the point of union of the cartilaginous with the osseous canals and along the suture lines. At the former point there would be not only the rounded eminence to receive special stimulation from any form of irritation within the canal itself, but the molecular stress exerted by such use of the man-

dible must have been considerable at this portion of the canal as well as along the unfused or weak tympano-mastoid and tympano-squamous, resulting in irritation of the more susceptible areas of the tympanic plate mentioned previously.

Referring once again to the literature on otosclerosis as being fruitful in its suggestions, I wish to quote from Gray's latest publication on the subject, taking the liberty of substituting the word "exostosis" for otosclerosos." He says—The other

No. 440. Chicama, Peru. An especially weakly fused suture, the tympano-mastoid. The entire tympanic plate is much thickened. (left).

general aspect of the problem is that the majority of individuals never do suffer from exostoses, no matter how badly or from whatever cause their general health becomes affected, or whether they suffer from some local disease of the ear, such as suppuration, middle ear catarrh, etc., or not. Now it appears to the writer that, viewed from this point of view, the logical conclusion to be drawn is that the cause of exostoses, that is the condition without which the disease cannot occur, is to be

found in the organ of hearing itself, and, further, that this condition exists in the organ of hearing of certain individuals, and in these individuals only. From this point of view, which the present writer believes to be the correct one, exostosis is idiopathic. That is to say, the disease occurs in people who have an inborn tendency to it, and that this is the only constant factor in the etiology. No doubt disturbances, either locally in the ears or constitutionally in the bodies of these individuals, may precipitate the onset of the disease, but no particular one of these disturbances need be presnt and in many cases they may all be absent. The single constant factor is the innate tendency to the disease and the fact that heredity influences are evident in many cases is strongly in support of this view." This "constant factor" can be postulated, in my opinion, from a consideration of the evolution of the organ of hearing. This process began with the primitive static labyrinth of the early fish and has ended with the complicated organ in man, both static and auditory. Can we not answer our problem by using the term "atavism"? Here in this disease is an attempt on the part of the organism, to return to its primitive state, an attempt to obliterate the hearing and the static mechanism in the reverse order of its development. This would be the natural order of retrogression, the latest developed being the first to suffer from the latent tendency or rather capacity of reversion that lurks in all forms of life, human or otherwise. The canals of our ancestors would be in the light of this fact, less stable than those of our immediate predecessors or fellow citizens. This may be regarded as one of the factors accounting for its much greater prevalence in primitive man. Those cases attributed to purulent processes in the middle ear and those in which the condition arises apparently spontaneously can be reconciled on this common ground. Why do not all cases of O. M. P. C. with or without polypi develop exostoses? The purulent process merely calls into action the latent tendency of the organism. The many other factors, rheumatism, syphilis, gout, etc., may act in the same manner. But underlying the whole process we can postulate the theory I have advanced. All the divergent clinical evidence that has been brought to light since the earliest discussions of the subject can thus be reconciled, i. e., the many etiological factors, the absence of any such, the hereditary element and the sporadic appearance in an individual with no apparent hereditary taint.

Summary and Conclusions.

Exostoses are relatively a rare condition in the aural canals at the present time.

They may exist along with some irritative condition in the ear, inflammatory or noninflammatory. Constitutional disturbances have also been found to exist coincidentally. These may all be absent and the condition linked with a hereditary, congenital, or developmental factor.

Practically all the exostoses found exist along the lines of union of the tympanic plate and the rest of the temporal bone. It is in these two regions that the bony canal is developed most rapidily from the annulus. It may be postulated that these ends of the annulus that proliferated the more rapidly are more susceptible to irritation of any sort.

An additional factor in the development of exostoses along the suture lines at these more susceptible areas, is the irritation due to mastication acting on what are in many instances weakly fused lines of union.

The case reported offers little light on the subject of the etiology except that excessive sea bathing may have been an exciting factor. The process microscopically and pathologically considered was a "proliferative periostitis". Accompanying the exostoses were an intumescent rhinitis, a markedly deviated septum, and an O. M. C. C.; conditions which, like those complicating an otosclerosis, may often be neglected or even overlooked. In all cases coexistent pathology in the head should be corrected. The general condition of the patient should be investigated at the time of examination and with regard to the circumstances at the onset of the aural symptoms. In this way many interesting and relevant facts may be uncovered.

Prehistorically considered, aural exostoses were more prevalent in primitive man. The etiological factor postulated in this regard is the excessive use of the teeth reacting on the more susceptible areas before mentioned (still more susceptible because of the earlier stage in evolution) producing lines of increased stress and even slight motion at the suture lines, very often weakly fused or largely unfused.

In view of the many causes given, we can conclude that the fundamental factor has not been discovered. I wish to advance the theory of atavism as a possible answer.

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THE PRESENT STATUS OF SKIAGRAPHIC INTERPRETATION AS AN ADJUNCT IN THE DIAGNOSIS OF CATARRHAL AFFECTIONS OF THE ACCESSORY SINUSES.

By ROSS HALL SKILLERN, M. D., Philadelphia, Pa.

I think most of us have long since as a routine procedure, sent our obscure sinus cases to the roentgenologist for his advice and have largely been guided by his report in our subsequent treatment of that particular case. If for example exudate was said to be present in the anterior ethmoid cells of a given side these cells were immediately opened even though direct examination had revealed nothing abnormal nor had the symptoms pointed particularly to those individual structures. How often at the time of operation have we been disappointed to note the absence of all pathological changes and it required a considerable stretch of the imagination to convince ourselves that the operation had really been indicated much less that the benefits which might accrue would bring about a complete or even partial relief of the symptoms. That we have been placing too great confidence in the X-ray reports to the detriment of our own better judgment has slowly been dawning on some of our minds for several years. Levy,[1] in an article on diagnosis of sinus disease states that the X-ray is overestimated. LaRue in a discusion of this paper says operation often shows positive disease while the X-rays even negative or even vice versa. Ballenger,[2] in an exhaustive study says a secondary place should be given to the X-ray and the main reliance should be placed on clinical means.

Boot[3] while discussing this subject said many of his reports had been absolutely wrong. Mithoeffer[4] says he considers the X-ray picture as an adjunct of secondary importance. Culp[5] cites a case in which a prominent roentgenologist after taking numerous negatives stated no trouble existed in the sinuses. Shortly afterward she was operated upon and both antra were found filled with cheesy matter and polypoid tissue degeneration. Chase[6] states it is possible to have pus present with practically no shadow on the plate. Guttman[7] on the other hand had a case which showed marked cloudiness of the antrum which on being

observed was found to be healthy. Dean[8] writes that radiography is very unreliable in infants and young children. Not so clear as in adults. The sphenoid and posterior ethmoids being almost always blurred on the plates. Long says the X-ray is of considerable service, etc.* All these opinions have been expressed within the past twenty-four months. It is my purpose to take these statements as a text for this paper and endeavor to present a frank and unprejudiced discussion of the actual value of X-ray plates to us as rhinologists as an aid to the diagnosis of inflammatory conditions within the sinus cavities.

Let us first briefly consider the standard methods of exposure which are instantly recognized, omitting those which present curious and bizarre shadows making the anatomical orientation difficult for the average laryngologist. The Frontal Sinus—(Caldwell)

1. Postero-Anterior. For Size and Shape. Shadow.
2. Lateral. For Depth and Thickness of Anterior Wall.

The Maxillary. Postero-Anterior for Shadow and Size.

2. Lateral. For Relation to Teeth.
3. Extended Chin (Waters and Waldron). Shadow for comparison with opposite side.

The Ethmoid.

1. Postero-Anterior. For Shadow and Extent.
2. Oblique (Rhese).
3. Stereoscopic. For Anatomical Configuration.

The Sphenoid.

1. Lateral. For Shadow as compared with ethmoid. Size and Shape.
2. Postero-Anterior Shadow.
3. Extended Chin. Shadow and Size.
4. From above, film in mouth (Pfahler) Shadow, Shape and Size.

These I think include the methods that today are in general use, the plates from which we are accustomed to examine and confirm the report of the roentgenologist. If we were asked which sinuses in their order were shadows more easily distinguished we would probably say: 1. Maxillary. 2. Frontal. 3. Sphenoid and 4. Ethmoid. This order would perhaps also

*Diveley leans quite to the opposite direction and states in 500 cases the X-ray alone made the diagnosis in 23% as these presented very little evidence. X-Ray Study of 500 Medical Cases for Paranasal Sinus Infection. Missouri State M. A., Jan. 1922.

check up with our clinical findings as far as confirmation was concerned. Why then do we have such divergent expressions of opinion as to the actual value of radiographs for diagnostic purposes and why have so many of us been disappointed after confirming our treatment to the X-ray disclosures. Sauer says that improper technic and faulty interpretations are usually the basis of its lack of efficiency and positive findings frequently depend upon the over-zealousness of the interpreter rather than upon the pathologic process. It is said the X-ray never lies. It shows what is there if one can judge by the super adjacent tissues. This to my mind is the crux of the entire situation. The shadows must be properly interpreted in order to be of value and as the personal equasion must enter largely into the readings, it is essential that the reader must have checked his experience with the subsequent surgery so that his statements as to findings will be reliable data for the rhinologist to unhesitatingly follow. This will undoubtedly occur when the rhinologist himself becomes an expert interpreter of roentgenograms. Before considering the individual sinuses let us for a moment diverge and discuss some of the common causes of errors in interpretation.

1. *Faulty position.* All of us at one time or another have seen ordinary photographs which could hardly be recognized by even a member of the family. Why cannot the same be true of an X-ray photograph? As a matter of fact, it may distort to such an extent that one receives a wrong impression of the plate with a consequently false interpretation. This can be laid to an error in technic and not the reading but would hardly occur in a well grouped series of exposures.

The presence of air in the sinuses appears to exert considerable influence upon the shadow as to sharpness of outline. Secretion in the sinuses even thick inspissated would appear to play a rather unimportant role although occasionally one could observe a line of demarcation at the fluid level. The shadow as a rule however is not greatly influenced. Swollen and inflamed mucosa gives a denser shadow than exudate and has often been confused with it.

Its relative value in the individual sinuses.

Maxillary. It is here that the X-ray as a diagnostic measure has its greatest value in sinuology. By means of the antero-posterior exposure comparison with the opposite side is easily and readily made and constitutes the basis of the interpretation which does not apply so well to any of the other sinuses, not

excepting the frontals. Even here however the possibilities of an error cannot be overlooked which may be caused by a shadow due to asymmetry of the bones of the face or the thickening of the lining mucosa in one of the antra the result of a former infection.

Frontal. Much information can be obtained from good skiagraphic plates provided the proper lines of exposure are observed. The postero-anterior for all around purposes is probably the best as it not only gives the shape, extent and position of the sinus but is very liable to give a fairly accurate reading of the condition of the sinus cavity. While shadows were occasionally observed in which no clinical evidence of inflammation was present, on the whole the plates furnished data which could be relied upon and coupled with the clinical symptoms were of considerable value in the subsequent treatment of the affection. The lateral position gives information as far as the anatomical configuration of the sinus is concerned such as its depth, the thickness and position of the anterior and posterior walls but throws little light on the contents or condition of the mucosa. Skiagraphs of the frontal sinus can then be considered of such value as to warrant them in all cases before operative procedures are applied.

Sphenoid. Opinions of both the sinuologist and roentgenologist seem to be converging in that while these sinuses are distinctly within the scope of the X-ray, it applies more particularly to the anatomical configuration and relations to surrounding parts and less to the actual pathological condition which obtains within the sinus cavities. The continual experimentation with new positions would seem to lend weight to this assertion.

Cushway[9] for example has tried all the various positions recommended and has been extensively experimenting in this direction but thus far the results have been disappointing. Long[10] in a comparatively recent writing considers it of considerable service in demonstrating the size and shape of the sphenoid sinuses but of limited assistance in giving information as to the gross pathology. As far as the positions of exposure are concerned there is little to choose between them, as long as there is no anatomical distortion which makes orientation difficult. The antero-posterior position bringing the sphenoid out through the orbital cavities seems to have the greatest vogue generally speaking but the oblique positions of Rhese and that of

Pfeiffer enjoy no little popularity, particularly in the larger cities of the East. None of these methods have proven satisfactory in my own work. I have never been quite certain that the operation would disclose the precise findings one had been led to expect and have usually not been disappointed in their expectations. Many times exudate has been positively diagnosed of which on operation no signs were visible and vice versa. I have long since discarded the roentgenogram as anything more than a possible confirmatory means to a diagnostic end. Bowen[11] a roentgenologist has come to the conclusion that neither the Pfeiffer position nor the reverse one is of much value in the diagnosis of sphenoidal sinus empyema. Let us hope the method recently devised by Pfahler[12] with the film held tightly against the soft palate and the rays directed downward may prove more reliable.

Ethmoid Labyrinth. It is here in affections of these cells where most help is needed from the skiagraph that we get the least. When I first had the opportunity to study skiagraphic stenoptican views of the ethmoid I felt that the X-ray had disclosed its hidden pathology and that hereafter it would be an open book. Subsequent experience has only added to my disappointment which seems to be shared by many of my colleagues. Thus Wherry[13] says the field of usefulness of the Ray as applied to the ethmoid heretofore has been most unsatisfactory. The reason for this is that a shadow or haziness in the ethmoid region does not always indicate an exudate or any change in the cells as an hypertrophied middle turbinate will give this appearance on the film. Purulent secretion in one or more of the cells will usually not influence the density of the shadow and even a thickening of the lining mucosa will scarcely affect it. The most reliable indication from a roentgenographic stand point for a pathologic process, is the blurring of the inter-cellular septa. If these cellular walls do not come out clear and sharp in several exposures undoubtedly a breaking down or a reabsorption has occurred which may be due to either a hyperplastic or suppurative process. If the absorptive process has caused the formation of one large cavity, the reading will of course be plain but we are not considering the simple cases but rather those that are more commonly met with. Summing up then the comparative efficiency in the various sinuses let us arbitrarily place the ratio of value about as follows:

Maxillary Sinus	85% efficient
Frontal Sinus	75% efficient
Sphenoid Sinus	40% efficient
Ethmoid Labyrinth	25% efficient

This does not mean that in a given number of cases this percentage will be found to be absolutely correct but rather the relative value of assistance the Ray gives in disclosing the pathological condition present in the individual sinuses.

In conclusion one must frankly state that the mere presence of a shadow in a sinus cannot be considered a reliable indication for operative intervention unless accompanied by the usual clinical symptoms pointing to disease in that particular sinus.

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DISCUSSION.

Dr. F. M. Law, New York said that the laryngologist and rhinologist should cooperate with the X-ray man in the interpretation of the plates. The X-ray is not always definite; lack of shadows leaves one where one started. The interpretation of the sinus plate will depend not alone upon the blocking out of pneumatic areas and presence of material, but also upon the condition of the bony wall of the cavity. Where there is thickened mucous membrane, there may be a bony change as

well. There will be a change of shadow in the floor of the antrum and separate ethmoid partitions. Changes are well marked in the old chronic cases, but the X-ray falls down in the acute cases. A better plate can be made after irrigation of the antrum. An opaque antrum shows polyp tissue. It requires education to properly interpret the plates. The glass plates show an apparently different result to the films. This must be taken into consideration.

Dr. Harold Hays, New York said that cooperation was necessary between the rhinologist and the X-ray man. He had his own laboratory but did not always agree as to the interpretation of the plates. One cannot rely on the plates in the absence of clinical signs. The X-ray man will say an ethmoiditis is on one side, when the signs are on the sis. Preliminary shrinking of the mucous membrane of the nose with other side. The X-ray man should not be allowed to make the diagnococain and adrenalin will procure a better, clearer X-ray picture.

Dr. F. N. Bigelow, Providence, R. I. said a method of projecting the sphenoid and ethmoid, against the odontoid process and their bony background, as in stereoscopic vision was very helpful. The clearness of detail gives a clue as to the technic and quality of the plate. If the outlines of the background are cloudy, the fault is in the technic of the plate. It is not enough merely to diagnose cloudy sinus. That is merely transillumination. By determining the increase in calcium deposit, one can get a more exact idea of the underlying pathology. The picture should be taken in the morning before the fluid in the cavity has drained out.

Dr. F. C. Ard, Plainfield, N. J. said that at the Killian clinic there was a nurse who used to do beautiful X-ray work. In cases where the plates showed no frontal sinus, operation proved this to be the case. It seemed to be possible to determine the presence or absence of the accessory sinuses by the plate. Later people began opening the sinus, solely on X-ray evidence. This is wrong; no sinus should be opened without clinical reasons. There should be close cooperation between the man who takes the plate and the man who knows the clinical picture.

Dr. W. B. Chamberlin, Cleveland O. said that this was the result of laziness on the part of the profession. The clinician has been too ready to turn the case over to the X-ray man to diagnose, and the radiographer has been too ready to seize upon the opportunity. It was refreshing to hear the liberal attitude taken by Dr. Law. Often one had a struggle, and had to advance clinical proof to get the X-ray man to admit he might be mistaken. The interpretation of the plate depends upon the comparison of various densities. There is a difference in the densities on either side of the skull.

Dr. Coffin (Chairman) said that no matter how much he admired Dr. Law's work, he would not take his diagnosis without seeing the picture, and the result of the conference was not always what they wished it to be. Transillumination is good (sometimes good for nothing). In one case (in Dr. Law's absence) the radiographer interpreted a plate, and stated the patient had no frontal sinus. The speaker said he made a different type of incision on this basis. He had to keep on cutting and cutting, half way up to the hair line and found an enormous diseased frontal sinus. On the other side a large sinus was indicated, and they went to do a Killian operation, and were fooled, because there was no sinus on that side.

COMPLICATIONS OF PARANASAL SINUS DISEASE IN INFANTS AND YOUNG CHILDREN.

By L. W. DEAN, M. D., Iowa City, Iowa.

The most serious result of paranasal sinus disease in infants and young children is death.

An infant two months of age came into our service in an extremely critical condition with a hemolytic streptococcic infection of paranasal sinuses and ears. The mastoids were drained with improvement. The paranasal sinus disease was treated. When the infant died the opinion was given that the cause of death was paranasal sinus disease. I was anxious to get the very best possible opinion about this case so the autopsy was held by Drs. Medlar and Armstrong, the two pathologists of the University Hospital, together. The mastoid wounds were clean. The superior and inferior meati of each side of the nose were filled with large accumulations of pus which seemed to come from the paranasal sinuses under pressure. In each maxillary sinus pus under great pressure was found. A complete general examination was made with negative findings. The pathologists expressed the opinion definitely that the cause of death was the paranasal sinus disease.

Another infant six weeks of age with a similar history died while it was under treatment for paranasal sinus disease of streptococcic origin. Both of these infants were very sick children when they came into our service. They were so sick that it was necessary to feed them by lavage. When an infant two or three months of age comes into our service with a temperature of 105½° due to paranasal sinus disease and we get the child early before the gastro-intestinal disturbances are marked the prognosis is very good. If when the treatment is begun the child has reached a stage where it will no longer take nourishment of itself the prognosis is very bad. This second infant who died, like the first, had pus under pressure in every paranasal sinus. The pathologist stated that this was the cause of death.

There may be an interdependence between chronic multiple infectious arthritis and paranasal sinus disease in infants and young children. This may be present at the age of three.

Case Reports.

On December 4, 1918, Baby E. T., age three, was referred to us by Dr. Steindler for examination for focus of infection in the upper respiratory tract. His diagnosis was chronic multiple infectious arthritis. We found chronic tonsillitis, adenoids, plus bilateral maxillary sinusitis. A hemolytic streptococcus was secured from both the maxillary sinuses and from the tonsils which, when injected into a rabbit caused arthritis. The tonsils and adenoids were removed and the diseased maxillary sinuses treated by simple irrigation with argyrol. This sinus disease apparently disappeared but, while the arthritis was improved, it persisted. Six months later an opening was made in each maxillary sinus through the antro-meatal wall and the sinuses treated. The sinus disease disappeared and with it the acute joint trouble. Two years after this on July 6 last, the mother of the little girl brought her into the Pediatric Service. The mother stated that since the little girl left the hospital two years ago she has been much improved; the first year she had apparently perfect health but last winter following a severe cold she had considerable nasal discharge. There was no trouble with arthritis at this time; two weeks ago she developed a stiff and swollen left ankle. Examination revealed pus in each maxillary sinus. A small opening was made in the antro-meatal wall and the maxillary sinuses irrigated; vaccines were used with an apparent disappearance of the maxillary sinus disease and of the acute manifestations of the arthritis. Several weeks ago I saw this child. She is perfectly well. She can dance and play as well as any child.

Naturally, when her sinus disease was being treated Dr. Steindler also gave the proper orthopedic treatment to the joint.

This case illustrates two things. First, that paranasal sinus disease may cause systemic infection at the age of three. Secondly, that paranasal sinus disease in infants and young children is sometimes not easy to permanently cure.

D. M., now nine years of age, is one of our most instructive cases. A brief resume of the case is as follows: when she was four years of age I removed her tonsils and adenoids with much improvement. That was in 1916 when we were not examining our four year old children for paranasal sinus disease. The little friend of ours would be today much better off if in 1916 we had examined, diagnosed, and treated the paranasal sinus disease which was undoubtedly present. When seven years of age we diagnosed a bilateral sphenoiditis and posterior ethmoiditis. The diseased condition was eradicated. The operation was followed by the most striking improvement in the arthritis, temperature, and general condition of the case. She was discharged in December 1919. In July, 1921, she returned with a recurrence of the acute arthritis. The sphenoidal and ethmoidal regions, much to my surprise, were perfectly healed and clean.

A careful examination showed pus in the left maxillary sinus. This sinus was drained and in three weeks there was a most noticeable change in our little patient. From a morose, disconsolate child, suffering from acute pain, she was changed into a happy child, free from pain. Her general appearance was wonderfully improved.

For six weeks before this little patient came into our service she received the most careful metabolic treatment and vaccines in the Pediatric Service

without improvement of her acute trouble. The only thing that gave this little girl relief from her acute symptoms when she was six, and when she was nine, was the treatment of her paranasal sinuses.

R. A. age four suffered from infectious arthritis involving the joints of fingers and toes. The joints were much swollen and painful. She could not walk. There was as yet no permanent deformity. When I saw her first she had bad tonsils and adenoids, plus maxillary sinusitis. Tonsils and adenoids were removed. Three months later she was much improved but still suffered with acute attacks of arthritis with pain and fever. The nasal sinuses were treated for one month. At the end of that time a streptococcus viridans persisted in each maxillary sinus and the arthritis remained active.

Then a small antro-meatal opening was made and the sinuses treated. This was followed by relief of the arthritis. Two weeks ago, that is sixteen months after she left our service, a report stated that she had been, and was, perfectly well.

W. F. is a case of recurrence of multiple infectious arthritis, that recently returned to our service from Dr. Byfield, Professor of Pediatrics, with the following note:

"My dear Dr. Dean:

One of the boys in the arthritis deformans series has been absolutely free from trouble since the operation which took place about two years ago, I believe. Just a few weeks ago he suddenly had a recurrence. The symptoms were pain in the joints with some swelling and stiffness, also some headache and an interesting change in disposition such as I would suspect would take place in sinus infection. I recall in this case that it was rather difficult to spot the infected area but it was finally located and the relief to the boy was the result. This boy has been unusually well since the operation, has had an excellent appetite and has done well in school. In the few weeks since the beginning of the recurrence he has lost weight and the appetite has been poor. The mother seems to feel that the treatment which you gave him was the thing which was responsible for his relatively long period of freedom from trouble and she is willing to have you go ahead again and not have any orthopedic treatment until the focal infection has been studied.

A. H. Byfield."

W. F. is now fourteen years of age. He came into our service at the age of eleven. His tonsils and adenoids were removed July 26, 1918. On September 25, 1918, because of the continuance of his acute joint infection, a second examination of his nose was made. A deflected septum with suppurative ethmoiditis was found. The examination of maxillary sinuses was negative. A submucous operation was performed on the septum and treatment instituted. At the end of two months' treatment a hemolytic streptococcus persisted in the posterior ethmoids and sphenoid of each side. These sinuses were very necrotic and on November 7 they were exenterated with the apparent disappearance of the sinus trouble the acute arthritis disappeared and returned only after a period of two years. Up to two weeks before the admission on June 29, 1921, he had been free from trouble. At that time knee and ankle joints became painful and swollen. Preceding this trouble he had had, as he said, a cold and a nasal discharge persisted.

The X-ray examination of the frontals and maxillary sinuses showed them to be quite clear. With the nasopharyngoscope pus was found in each

posterior ethmoidal and sphenoidal region. The left sphenoid was particularly badly diseased. The upper posterior sinuses were again operated and post-operative treatment instituted. As the result, the nasal discharge is rapidly disappearing and the joints are becoming rapidly better. This case illustrates nicely the fact that when the removal of tonsils and adenoids in a child does not remove the focus of infection, that the paranasal sinuses should be carefully examined. It also brings to my mind again, forcibly, the fact that in some cases a cure of paranasal sinus disease in children is very difficult.

Bronchitis is a very common result of paranasal sinus disease in infants and young children. In short I think that every infant that has bronchitis, even if adenoids are present, should be suspected of having paranasal sinus disease and the paranasal sinuses should be examined.

Paranasal sinus disease may be the source of infection for pneumonia.

Baby J. S. three and one-half months of age was under our care for suppuration of the paranasal sinuses plus mastoid infection. Each mastoid had been operated and the mastoid wounds were perfectly clean but not yet healed when the baby developed pneumonia. At the time we were treating the paranasal sinus disease. The baby died on February 1, 1922, and the autopsy is as follows:

The right lung is crepitant throughout but shows no area of consolidation. The left lung is covered by a thick layer of fibro-purulent material. The lung is airless, firm, but of a dark color. On cut sections the entire organ is of a deep red color, firm and fairly dry. There is a small amount of free fluid which can be squeezed out of the bronchioles. The whole lung presents a fairly typical picture of broncho-pneumonia.

Nasal cavity. The nasal cavity was opened by a lateral incision through the base of the skull which permitted the halves of the skull to be quite widely separated. The walls of the entire nasal cavity were bathed in pus. Smears were made from this pus and microscopic examination revealed many pneumococci. The pus was particularly thick and abundant in the superior meatus. A probe pushed backward through the superior meatus on either side entered a cavity approximately 6 mm. in diameter which lay partially within the body of the sphenoid and was filled with thick creamy pus. The superior turbinates were cut away and the posterior ethmoids exposed. These cells were also filled with pus. The middle and inferior turbinates were then removed and the lateral walls of the maxillary sinuses exposed and removed. The sinuses were about the size of a navy bean and were filled with pus which swelled up when the wall was removed as if it were under pressure. There was no macroscopic evidence of ulceration of the mucosa or osteomyelitis. The mucosa of the sinuses was pale and edematous. Dr. Armstrong autopsied the head. Dr. Medlar, head of the Department of Pathology and Bacteriology of the University of Iowa was present and both he and Dr. Armstrong expressed the opinion that the source of infection for the pneumonia was the paranasal sinus disease.

There is frequently an interdependence between paranasal sinus disease in young children and asthma. The following two letters from Dr. Byfield, head of the Department of Pediatrics in the University of Iowa, have some bearing on this subject:

"Dear Doctor Dean:

You might be interested to know that O——— S———, aged eleven years, has been returned to our service for observation. This patient has had asthma for nine years, the attacks coming on at intervals of about a week previous to his first admission to this service. He received nasal sinus treatment from you and with the exception ot a very mild attack of asthma a day after his return home, he has been free from every evidence of the disease. He has been able to go to school and has lost a lot of his shortness of breath. These statements have been obtained from the patient himself and are, as far as we can tell, reliable.

Albert H. Byfield."

"Dear Dr. Dean:

We have just heard from the parents of eight year old R. S. and twelve year old E. M., in reply to circular letters. Each one of these children had one mild attack of asthma a few days after leaving the hospital and none since that time. This makes the third case in which satisfactory results have been obtained.

Albert H. Byfield."

One of the characteristics of sinus disease in young children is headache; usually frontal and interpreted by the parents as being due to eye strain. We may have the neuralgic type from involvement of the nasal ganglion. Sometimes the frontal pain is very excruciating. It is interesting to note that occasionally a tuberculous meningitis beginning will cause the same type of pain as an acute frontal empyema. In acute paranasal sinus disease the patient also complains usually of nasal stoppage or a feeling of fullness in the head.

Infants do not complain of headache. Dr. Byfield is of the opinion that a child complains of headache first in the neighborhood of from five to seven years. He reports that in Dr. Still's text book in discussing the migraine of childhood he mentions that at the age of five years is when the first complaint of headache is usually made.

The following case is very interesting. Mrs. S., age 44, came complaining of right sided headaches beginning at the age of six years. The headaches have always been of this type. Many times a diagnosis of migrain has been made and she assumed that this was a headache which was to remain with her for life. In examining the paranasal sinuses when a maxillary sinus was irrigated the patient immediately said that the pressure caused the typical headache. Eradication of a chronic maxillary sinus disease resulted in an

immediate and permanent disappearance of a headache which had persisted for thirty-eight years.

In children, fever usually accompanies acute paranasal sinus disease. In infants the fever is usually very high, 103° to 105½° with daily remissions. It is axiomatic, I believe, with Pediatrists that in all cases of fever in infants where the cause is not definitely known, the ears should be examined. As I view it, the examination of the paranasal sinuses is of equal importance. The fact that an acute ear is present does not exclude acute paranasal sinus disease and the individual who has in his care an infant with an acute suppurative otitis media in which after myringotomy fever persists, and who does not make an examination of the paranasal sinuses is liable to make a serious mistake. I have seen several cases where mastoidectomy had been recommended in infants where the cause of the fever was infection of the paranasal sinuses and the fever disappeared with the treatment of the paranasal sinuses.

Recurrent fever in infants and young children without apparent cause is sometimes due to disease of the paranasal sinuses.

Three years ago, I. H., then seven years of age, was referred to us by Dr. Byfield. He gave a history of attacks of sudden elevation of temperature of about 102° without apparent cause. During the attacks of fever the boy was not ill. He seemed slightly indisposed and the fever was found by the use of the thermometer. This condition had begun at about the age of two. It had been made better by the removal of the tonsils and adenoids one year before but persisted. Three years ago the paranasal sinus disease which was present was eradicated and his trouble has completely disappeared.

Periodic vomiting may be associated with disease of the paranasal sinuses.

F. M., six years of age, came into our service, suffering from attacks of recurring fever with vomiting. These attacks are complicated by acidosis. The eradication of an ethmoiditis resulted in a disappearance of the attacks of vomiting and fever.

The work on the paranasal sinuses was done almost two years ago. Three months ago a report from the boy stated that he had not had any trouble up to that time.

The following is the report of a case of periodic vomiting that was carefully studied by us and in which the result was disappointing.

L. D., age 19 years, entered our service with the following history:

Since the age of two he had suffered from periodic vomiting. There is one attack every two or four weeks. The internists are of the opinion that at this time there is a decided functional element in the case. The first attack at the age of two and every one since has the following history: Cold in the head, followed by dull headache in frontal region and top of the head, vomiting. Chronic suppuration of all the paranasal sinuses except the left frontal was found. All sinuses have been drained. The number of attacks of vomiting have been greatly decreased but he still has an occasional very

severe attack. We cannot help but feel that in the beginning this was periodic vomiting due to paranasal sinus disease, and that later a functional element appeared in the case so that the eradication of the paranasal sinus disease did not result in a complete cure.

A most common complication of paranasal sinus disease especially in infants and young children is gastro-intestinal disturbance. This is usually so severe that it is thought to be the primary trouble. As to whether this disturbance is the result of the swallowing of the nasal discharge or the result of the elimination of bacterial products through the gastro-intestinal tract, I do not know. Dr. Byfield suspects that the milder cases at least are the result of the ingestion of the purulent discharges and that in the more severe cases, just as we may get an acute nephritis from the elimination of bacterial products, we have an acute gastro-enteritis.

Dr. Scrutton (Laryn. January 22, page 70) says that gastro-intestinal troubles in acute otitis media are due to the ingestion of the purulent discharge from the ethmoids. This purulent discharge is also the cause of the purulent otitis. I do not think we can emphasize too strongly this point made by Dr. Scrutton. We must not assume that the systemic disturbances of an acute otitis in infants are always the result of the ear lesion because they are so frequently the result of a lesion of the paranasal sinuses.

Pyelitis may be related to paranasal sinus disease just as it is related to chronic infection of lymphatic tissue in the naso-pharynx. In a personal communication Dr. Stucky told me that he had several such cases. We have had two where the elimination of the paranasal sinus disease seemed to be the thing which was responsible for the disappearance of the pyelitis.

Acute cervical adenitis in infants and young children may be the result of paranasal sinus disease.

N. N., seven years old, suffered from very extensive acute cervical adenitis with temperature of 105°. (X-ray showed well developed frontals. Frontals, anterior ethmoidal and maxillary sinuses blurred.) Tonsils and adenoids out one year previously. This boy was very ill. Treatment of diseased maxillary sinuses alone resulted in a disappearance of his trouble in one week's time. I have seen an infant one year of age with the same condition the result of ethmoiditis, relieved by the treatment of the ethmoiditis.

Recently, Renaud, a French physician (International Medical and Surgical Survey, December 21, 88b/-348) expresses the opinion that the cause of death in infants who are supposed to die from gastro-intestinal disturbance is frequently a suppuration of the mastoid. Our experience is that the suppuration of

the mastoid in infants is frequently secondary to the paranasal sinus disease.

I trust that I am not infringing on the province of the pediatrist when I express the opinion that if the paranasal sinuses and mastoid cells are carefully autopsied in all infants who died that a surprisingly large number of acute suppurative infections will be found present in these cavities.

Certainly paranasal sinus disease is frequently the source of infection for acute otitis media. In the infant ward this winter we had an epidemic of acute suppurative otitis media. Every one of the children with infected ears had an acute infection of the paranasal sinuses. When drainage of the ear did not relieve the high temperature, treatment of the paranasal sinuses frequently did. At least four infants died, in each of the four there was pus under pressure in the paranasal sinuses.

In the obstetrical service we had a very interesting case. Baby C., seven days old; at the time of birth the mother had an acute paranasal sinus disease with an acute ear. We secured a hemolytic streptococci from both paranasal sinuses and ear. When seven days old the baby developed fever. Examinations showed suppurative ethmoids, acute otitis media bilateral. Both nose and ears were treated. Later a mastoidectomy on each side was performed. The baby gradually failed and when two months old died. Autopsy performed by Dr. Medlar, head of the Department of Pathology in the University.

Findings: Little pus in each tympanum; ethmoidal and maxillary sinuses filled with thick pus under pressure. Cause of death paranasal sinusitis and suppurative otitis.

Paranasal sinus disease may cause in children, but not in infants, brain abscess.

Paranasal sinus disease may cause meningitis at any age.

Dr. A. G. Andrews of Chicago (Ill. Med. Jounr. Nov. 1921) reports a child six years of age dead from meningitis. At autopsy the meninges were found involved from necrosis of the roof in the sphenoids. The sphenoid was filled with pus and granulation tissue. No examination of the nose had been made before death.

The ocular complications of paranasal sinus disease in infants and young children are most important. There is frequently an interdependence between the infection of the sinuses and phlyctenular conjunctivitis.

Turner, in discussing the etiology of phlyctenular conjunctivitis (Amer. J. of Oph. Feb. 10) is of the opinion that ethmoids are the important etiological factor in phlyctenular conjunctivitis. He is of the opinion that when improvement of the eyes

follows the removal of tonsils and adenoids, it is due to the removal of the latter's influence on the ethmoids, which are present, that results in the improvement of the eyes.

S. B., a colored boy aged ten years who was referred to us because of phlyctenular conjunctivitis. This boy was in the hospital for six months. When he first entered the hospital a bilateral maxillary sinus disease which he had was treated and eradicated. The usual local and general treatment for phlyctenular conjunctivitis was given by Dr. Boiler, the ophthalmologist. At the end of his six months of treatment his nose seemed to be perfectly clean but the corneae remained injected and photophobia was marked. Dr. Boiler reported that the eyes had remained absolutely stationary for six months. The paranasal sinuses were cultured and from the posterior ethmoids and sphenoid on each side a streptococcus hemolyticus was found. The posterior ethmoids and sphenoid were drained. Seven days after the drainage of the posterior ethmoids and the sphenoid the eyes were very much better and in three weeks all acute trouble and photophobia had disappeared.

L. G., a little child nineteen months of age, suffered from phlyctenular conjunctivitis which disappeared with the disappearance of a paranasal sinus disease and reappeared with the nasal trouble.

A. L., a boy age two and one-half years of age, suffered from phlyctenular conjunctivitis. This boy had bone tuberculosis and tuberculosis of the pharyngeal tonsil plus a non-tuberculous maxillary sinus disease. The etiological factor in the paranasal sinus disease was pneumococcus type 4. The treatment of maxillary sinuses and the injection of a vaccine prepared from the sinus discharge was of much benefit to the phlyctenular conjunctivitis. This case is interesting because the treatment of paranasal sinus disease in a patient manifestly suffering from tuberculosis was an aid in the treatment of phlyctenular conjunctivitis.

Retrobulbar neuritis is an occasional complication of paranasal sinus disease in children.

Dr. McCullough (Trans. N. Y. Acad. Med. October 26, '21) reports that in one year he saw three cases of retrobulbar neuritis secondary to paranasal sinus disease in children under fourteen years of age.

Dr. J. B. Gregg recently sent me the report of a case of retrobulbar neuritis in a child that was eradicated by the treatment of the diseased posterior ethmoidal and sphenoidal cells.

White (Laryn. Aug. '21) reports several cases of involvement of the optic nerve in children five years of age and older.

Orbital cellulitis may result from paranasal sinus disease in infants and young children.

Baby B. O., eighteen months of age, had orbital cellulitis, secondary to maxillary sinusitis and ethmoiditis. The orbital wall of the ethmoid was broken down and the neighboring bones were also involved.

P. B., aged four, had an acute pan-sinusitis suppurative with orbital cellulitis on the same side. We made an opening in the maxillary sinus. The use of

suction, nasal irrigation and argyrol in the nose resulted in the disappearance of the orbital lesion.

Baby H., 6 months of age, was in our service with a diagnosis of maxillary sinus disease secondary to posterior cleft, suppurative otitis media had developed. While the patient was under treatment for mastoid involvement an orbital cellulitis with abscess of the cheek formed. It is interesting to note that in this six-months old baby, the existence of the maxillary sinus disease had been proven before the appearance of the orbital cellulitis.

DISCUSSION

Dr. W. V. Mullin, Colorado Springs, said that different parts of the country evidently produced different types of infection, according to the climate. He had seen a series with bronchitis. The sinus disease can be cured in these, but the bronchitis is there to stay. The disease is seen by the internist first, and he must see that the disease is stopped in childhood. They are usually diagnosed tuberculosis. The internist and pediatrician must be trained to refer these cases to the rhinologist for diagnosis.

Dr. Coffin, New York City, said he had worked on these cases for years, but did not see such young children as Dr. Dean. He had some interesting cases from 9 to 11 years of age. The symptoms and complications were the same in adult life as in childhood. One girl was being treated by an ophthalmologist for blindness, then she was X-rayed for brain tumor. With attention to the sinuses, vision cleared up to 20—40. Another girl had vomiting and convulsions, she looked septic and anemic. With treatment of the sinuses the girl got well and gained 25 lbs. in weight. If the pediatricians were able to treat these cases, as they take out tonsils, they would get well quicker, but they are slow in referring them to the nose men.

Dr. Harris P. Mosher, Boston, Mass., asked how Dr. Dean could demonstrate pus under pressure on the cadaver.

Dr. F. P. Emerson, Boston, Mass., said that Dr. Dean's paper was interesting and instructive. The further back one could go in tracing a pyogenic focus, the better. Dr. Emerson found that the X-ray gives positive evidence of sinus disease in 80 per cent of cases. In two cases he had noted pan-sinusitis on the side of the infected ear.

Dr. F. M. Law, New York City, said that it is difficult to keep young children quiet enough to get a clear X-ray. The plate must be perfect to show the pathology. They found that in eye cases at the Manhattan, there was not a single case of conjunctivitis or purulent condition that there was not some evidence in one or all the sinuses of disease.

Dr. Dean (closing) said that they were not dealing with operative treatment of the paranasal sinuses. It would be impossible in infants and young children. In regard to Dr. Mosher's question, perhaps he was wrong, but he meant to say that when the maxillary sinuses were opened the pus welled up like a spring. He would advise looking for the trouble in the paranasal sinuses in these young children and taking the proper care of them.

THE IMPORTANCE (1) OF THE ABSENCE OF REACTIBILITY OF THE VERTICAL CANALS OF BOTH EARS TO STIMULATION BY THE COLD CALORIC IN THE DIAGNOSIS OF INCREASED INTRACRANIAL PRESSURE OF THE POSTERIOR FOSSA, AND (2) OF THE REDUCTION OF THE DURATION OF THE NYSTAGMUS FROM TURNING IN DIFFUSE LESIONS OF THE CEREBROSPINAL SYSTEM PATHWAYS OVER THE CEREBELLAR CORTEX (AS IN PROTECTIVE MENINGITIS AND LINEAR FRACTURES OF THE SKULL OF THE POSTERIOR FOSSA).*

By WELLS P. EAGLETON, M. D., Newark, N. J.

Synopsis.

I. Technic:

A department of Neuro-otology under the direction of an otologist, but largely operated by a trained technician, is advised in all hospitals.

II. Auditory Apparatus:

(a) During the evolution of life the auditory apparatus was a comparatively late development: it is a very highly specialized function.

(b) The accepted hearing tests are largely valueless for differential diagnostic purposes as far as the central portion of the auditory apparatus is concerned.

III. Vestibular Apparatus:

(1) A vestibular apparatus for orientation is well developed in very low forms of invertebrate life: it is one of the earliest of the special senses. Because of its early development its function has become largely an inherent property of the nervous tissue. It is doubtful whether any one special central tract is indispensable to the performance of orientation. Consequently, injury to or destruction of a vestibular tract or pathway is quickly and at times completely compensated for.

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Clinical Application:

Because of these biological facts a fundamental mistake has been made in trying to formulate rules only applicable when dealing with highly specialized tracts such as are present in the speech mechanism:

(1) Because of the low and primarily reflex (automatic) activity of both the vestibular mechanism within the cerebellum, compensation is easily established; consequently, gross surgical lesions affecting the central vestibular apparatus, if of slow growth, may produce either very transient spontaneous symptoms, or, in chronic lesions, no symptoms whatever.

(2) For the same biological reason, in interpreting the findings of the vestibular tests, the presence or absence of the following factors should always be considered, as they modify the vestibular manifestations:

(a) Is the lesion microscopic or diffuse?

(b) Has compensation had time to be established, and, consequently, is the lesion acute or chronic?

(c) Is increased intracranial pressure present?

(3) In microscopic lesions acutely destructive of a so-called "vestibular pathway" not associated with increased intracranial pressure, exact localization by means of the vestibular manifestations alone frequently is possible; as in minute hemorrhages, disseminated sclerosis and syringomyelitis.

(4) The vestibular manifestations of increased intracranial pressure, viz., the reduction of the reactibility of the vertical canals of both ears to the cold caloric (the reduction of the opposite side in the author's opinion, being the greater) is of the greatest clinical value.

(5) In surgical lesions, because of the rapid and more or less complete establishment of compensation and the overshadowing vestibular manifestations of increased intracranial pressure, localization of lesions by an examination of the vestibular apparatus alone is difficult, and generally impossible.

(6) Certain localized cerebellar surgical lesions, such as acoustic tumors, frequently can be diagnosed by the vestibular tests alone. The vestibular manifestations in such cases are a combination (1) of the effect of direct pressure on the vestibular tracts between the labyrinth and the brain stem as shown by the homolateral deafness, the absence of reactibility of all the homolateral canals, the spontaneous falling, and (2) the effect of the increased intracranial pressure of the posterior fossa in abolishing the reactibility of the vertical canal of the opposite side.

(7) In surgical intracerebellar lesions, such as gliomas or diffuse suppurative lesions (abscesses), localizing evidence may or may not be obtainable from the vestibular reactions; but the findings frequently are so transient and changeable, and as yet so imperfectly understood, that localizing surgical diagnosis can rarely be fundamentally based upon the findings of the vestibular tests alone.

(8) However, at any moment, due to an invasion of an intracerebral portion of the vestibular apparatus, spontaneous or induced, vestibular manifestations may appear, of localizing value—spontaneous vertical nystagmus, lateral deviation of the eyes—all of which usually rapidly disappear because of compensation.

(9) In exceptional cases, the spontaneous vestibular manifestations continue for indefinite causes probably due to invasion of, but without destruction of, the vestibular nuclei in the brain stem.

Clinical Applications:

(10) The immediate reduction of the duration of the nystagmus obtainable from stimulating the horizontal semi-circular canals by turning, is of distinct surgical and medico-legal value in diagnosing certain diffuse lesions of the posterior fossa. In the author's experience, this occurs in those lesions which are associated with a blockage of the cerebrospinal fluid pathways by exudate, such as in protective meningitis or by blood in fracture of the skull, chiefly of the posterior fossa.

Conclusions:

(11) These two vestibular manifestations, viz., of increased intracranial pressure of the posterior fossa, and of blockage of the cerebrospinal fluid system pathways of the posterior fossa, should always be considered in all attempts at localization of surgical lesions of the posterior fossa. In the author's experience they are the two chief intracranial vestibular manifestations constantly present so as to be of consistent practical diagnostic value in cerebral surgery.

There is clinical evidence that sudden invasion of the frontal lobe causes spontaneous past-pointing. This rapidly disappears. There is also clinical evidence that too sudden invasion of the temporal lobe causes loss of quick component in induced nystagmus by application of the cold caloric applied to the side of the lesion. This also rapidly disappears.

There is clinical evidence that sudden involvement of the cerebellum will cause nystagmus or a lateral deviation of the eyes. Both of these also rapidly disappear although in certain lesions, such as tumor and irreparable damage to the cerebellar nuclei the nystagmus persists.

VESTIBULAR AND AUDITORY TESTS IN INTRACANIAL SURGERY.

I. Technic of Otological Examinations in General.

At present the routine aural examinations in our large hospitals, if conducted at all, consist of an inspection of the drum membrane and a test to ascertain if the patient hears with each ear separately. Yet, in every general hospital there is constantly a mass of material which, if a systematic effort were made to correlate it, would throw light on this complicated subject.

In my opinion, all cases that enter the surgical wards for cranial injuries should have during treatment or at least before leaving the hospital, a routine otological examination by the functional tests. The same should apply to all cases in the medical wards which complain of vertigo, tinnitus or deafness; while, of course, no neurological examination should be regarded as complete without containing a full record of the vestibular reactions, both spontaneous and induced.

To make this possible there should be established in all general hospitals a department in charge of an otologist, although in my opinion the techical part of the work should not be required of him personally. All routine technical detail should be delegated to a male or female technician of intelligence. It is asking too much of the otologist to require him to devote the large amount of time necessary for the tests, with—in the majority of cases—negative findings. It would be as reasonable to ask the attending physician or surgeon to perform the routine urine examination.

The technician should be instructed by the otologist how to conduct the tuning fork tests; how to measure the duration of the bone conductions and to perform calorics on the vertical and horizontal canals. His report should be turned over to the otologist, and from it the otologist should make his diagnosis, verifying all abnormal findings. A few hours devoted to the instruction of such a technician in the use of the turning chair and the cold caloric—at first on a series of normal cases, followed later by abnormal ones—would soon make this possible. For the chief requisite is not medical knowledge, but keen powers of observation, associated with patience in execution, truthfulness in recording, and enthusiam for the work. Until this division of the technical work is generally established, we shall make little progress in the vestibular tests.

The above is put forward, not as a suggestion, but as a special plea, because work so conducted has been admirably successful in the two instances in which I have had authority to enforce it.

II. Audition Tests in Cerebral Localization.

Evolutionary Facts of Clinical Value:

Audition is one of the late developmental additions in the evolutionary ascent of life. It is by means of the auditory function that speech was evolved, and from speech have come all the still higher psychic attributes possessed by man. Conse-

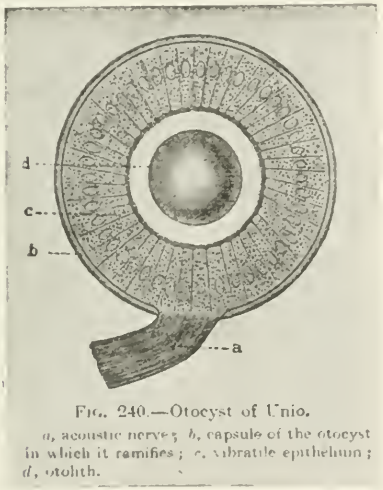


Fig. 240.—Otocyst of Unio.

a, acoustic nerve; *b*, capsule of the otocyst in which it ramifies; *c*, vibratile epithelium; *d*, otolith.

Figure 1. Vestibular Apparatus In Bivalve (River Mussel) Showing Complete Vestibular But No Auditory Apparatus.
From Morat, J. P.: Physiology of the Nervous System. Keener, Chicago, 1906.

quently, the auditory apparatus, both central and peripheral, is highly specialized—higher even than the visual apparatus. In man one of the evidences of this is that, although there is a hearing centre situated in both hemispheres, the auditory apparatus is the only one of the special senses, outside of speech, which in certain parts *still has a tendency* to a cortical centre localized in but one cerebral hemisphere—the left; for while destruction of the hearing centre in the horizontal gyrus (Hershel's gyrus) of one temporosphenoidal lobe does not cause deafness, in certain cases injury to this gyrus on the left side will occasion word deafness.

All the other senses have in the evolution of life become represented equally in the brain's cortex of both hemispheres.

An auditory apparatus is only present in the comparatively higher forms of life, in contradistinction to the vestibular apparatus which is well developed in very low types. While an otolite (with neuro-epithelial cells, the primitive part of the vestibular apparatus) is present in a very low form of invertebrate (in one of the low sub-divisions of the coelenterata), and the primitive medusa (jelly fish) has a well developed vestibular apparatus ("an otocystic formation with ciliated cells connected by nerves to a nerve ganglion, and this, in turn, with muscles"*), there is no evidence of a hearing apparatus in the invertebrate kingdom. It is only among the vertebrates, in which the vestibular apparatus is fully developed, that a hearing apparatus first appears. From these rudimentary beginnings it is gradually developed up to the mammals and becomes complete in man. Fish have no cochlea, although possessing a well developed vestibular apparatus with a utricle and three semicircular canals. Snakes have a rudimentary cochlea (reduced to a quarter of a turn). "In birds the cochlea generally remains rudimentary, but the semicircular canals are highly developed."*

This comparatively late acquisition by animal life of this highly specialized sense, audition, explains the clinical facts:

(1) that on account of its high specialization slight injuries are apt to destroy it totally;

(2) that injuries to the apparatus are slowly or never compensated for;

(3) that it is also because of its high specialization that we have as yet no proper diagnostic means of *localizing central lesions of the auditory apparatus* outside of the clinical appearance of word deafness.

We must realize that hearing is not only a highly specialized function, but it is an intellectual—psychic—function above the reflex-subconscious sphere, in which the vestibular function fundamentally resides. To hear and interpret, we must *listen.*

Audition, however, has an intimate association with the subconscious.

A cat, after the removal of the cerebral hemispheres, striata and thalami, shows orientated movements of the pinna and neck in response to sounds, such as a bark, whistling, et cetera.

*Morat, J. P.: Physiology of the Nervous System; English Edition, 1906, p. 601.

A protective reaction by the auditory motor is the jump reflex—showing how low down in the subconsciousness is the association between the reaction to sounds and orientation, the chief end organ of the latter being the vestibular apparatus.*[1] But the decerebrated cat does not hear in the higher sense in which the patient values hearing, for the purpose of intercourse with his fellows.

To hear speech we must listen, and this necessitates a motor and an intellectual effort. If we do not listen, in church, for instance, the speaker's voice soon fades away as well as the text about which he is discoursing.*[2]

Tests for Localizing Lesions of the Auditory Apparatus.

Although the violin for a musical instrument is the nearest approach to the human voice, in reality it bears no direct relation to it; in fact, no musical instrument can be adapted to act as a standard for speech, for the auditory apparatus of man, both peripheral and central, is especially specialized to perceive and interpret the human voice. From the viewpoint of the patient and from a proper diagnostic standpoint, it matters little how well or how poorly musical tones are heard if the modulations of the human voice are not correctly perceived.

But by some mischance the tuning fork, which furnishes a standard of *musical tones*, was adopted to differentiate the extent and position of lesions of the auditory apparatus.

It will require a much more delicate apparatus, based on a totally different principle, before we shall be able to diagnose the exact location of the lesions in the central portion of the auditory apparatus; for speech is made up of considerably more than a narrow range of musical notes.

Of the large number of musical instruments that have been put on the market as aids to *differential diagnosis of the site of the lesion, whether peripheral or central*, I should say (from a long personal experience with the following—Hartman's Set of Tuning Forks, Edelman's Forks, Gradenigo's Visual Forks, Koenig's rods, Politzer and Hays' Acumeters, and Galton's Whistle) that they are all *largely overrated*. Whatever practical information may be useful in differentiation is obtainable from one fork of long duration, excepting possibly in the case of partially deaf mutes, with whom my experience is so limited that I am not qualified to speak.

*1—Sherrington, C. S. and Forbes, A.: American Journal of Physiology, 1914, Vol. XXV, p. 367

*2—"The most satisfactory theory is that in the act of listening the resistance at the various synapses in the auditory path becomes diminished by some such process as a throwing out of dendrites, which brings those of contiguous neurones into more intimate connection. In inattention the synapses are unswitched, the resistance being increased by the retraction of the dendrites." Hurst, Arthur F.: "Psychology of the Special Senses—Hearing, Listening, and Hysterical Deafness." The Lancet, August 7, 1920, p. 285

On account of the great difference in the personal equation between different patients and observers, it is necessary that a tuning fork of over 200 d. v. per sec., be used uniformly to obtain even the meagre diagnostic information that the tuning fork may furnish.

III. The Practical Diagnostic Value—General and Localizing of the Vestibular Reactions in Cerebral Surgery.*

Present State of Knowledge:

The vestibular reactions of the end-organ—labyrinth—involvement are now fairly well understood, although many of the views of the physiology and pathology of the labyrinth itself are entirely hypothetical and are constantly being modified.

The anatomical and physiological knowledge of the cerebral portion of the vestibular apparatus is, however, still very imperfect.

Following the initial and enthusiastic acceptance of the localizing assistance of the vestibular tests in intracranial lesions, their practical value is to-day being seriously questioned. We must frankly admit that the subject is still in considerable confusion, and many of the findings are as yet unexplainable.

Disastrous Results of Hypothetical Teaching, not Based on Clinical or Experimenting Experience:

An injury has been done and a proper study of the tests retarded by the publication as facts of what in reality are but hypothetical deductions. Undue emphasis has been placed upon selected and isolated cases in which microscopic lesions of the cerebellar peduncles and of the posterior longitudinal bundle have been successfully diagnosed.

The positiveness of the statements created the impression that the tests, if properly performed and interpreted (from the viewpoint of adequate anatomical and physiological knowledge of the nervous system) are so accurate that a diagnosis of the localization of lesions of the medulla, pons, and parts of the cerebellum may be uniformly made (although anatomically a large part of these "tracts" still are problematical); the inference, of course, being that when such a diagnosis is not forthcoming the fault lies with the observer and not with the tests themselves.

There is no doubt that in very exceptional cases in microscopic lesions, such localizing diagnosis is possible; but the publication of a few isolated, brilliant diagnoses has discouraged the independent worker, because, failing to obtain localizing assistance

*Read in part before the American Otological, Rhinological and Laryngological Society at its Annual Meeting at Atlantic City, June 1, 1921.

in the average *surgical* case, he naturally has regarded the fault as with himself—in his lack of ability to interpret the findings correctly. The effect has been to intimidate the otologists, make skeptical the neurologists and neuro-surgeons, and, in consequence, to discourage a continuation of the work.

Fallacy of Attempting to Apply Theroretic Physiological Deduction to Surgical Lesions:

In those books in which few or no references to individual work are made, the brain is regarded as an inert body—a sort of complicated system of telephone wires, through which it is possible to prevent such and such a transmission "by interference" with such and such a tract without disturbing the other tracts: much as a lineman "cuts out" a wire.

It appeared so logical that interference with a pathway should cause loss of this or that part of a reaction that for a long time (in common with the rest of the profession) I failed to perceive the fundamental defect in the application of these theories to surgical diagnosis.

In the final analysis, all real scientific progress must rest on the accumulated experience of many. Theoretical considerations, which are not based on and fully verified by experience, tend, by misleading, to retard a proper understanding of disease. Some of these postulates are undoubtedly true, but as always occurs in hypotheses not founded on experience, in these publications (one copying from another) certain factors present in surgical lesions of the brain were disregarded, viz., increased intracranial pressure, the acuteness and nature of the lesion, and the influence of the establishment of compensation.

All these factors fundamentally modify the objective reactions of the functional action of the brain—which the vestibular reactions primarily are.

Prior to 1914, having devoted considerable attention to the vestibular tests and having become somewhat discouraged by the diversity of the findings, I was not only impressed by these published accounts as truths by the facility with which they apparently clarified so many complicated problems, but also convinced that my failures were due to my inadequate knowledge. Not realizing their fundamental weakness, I abandoned my pre-conceived opinions, the result of experience, and adopted the published statements as demonstrated facts, only to find after many years that the statements were in many cases fallacious.

This has been the general effect upon the profession, as an examination of the literature of the past few years will demonstrate; for not only has little been written on the subject, but what has appeared deals in generalities of the most elementary nature. More significant still is the fact that while the reports of cerebro-surgical cases of brain tumors have enormously increased, few of them contain detailed records of the vestibular reactions; which forces the inference that the vestibular tests in cerebro-surgical conditions either have been generally abandoned, or have been of so little assistance as not to warrant their incorporation in the published report.

Physiological Considerations:

The vestibular reactions are, as we have seen, in many particulars very primitive reflexes of orientation, injury to which is followed by compensation for the reason that the more primitive the reflex the more diffuse and varied are the ways by which such impulses are transmitted; the reflex action being assumed by the nervous tissue as a whole. Thus, a man with a paralyzed arm from cortical destruction still gives a muscular twitch when a primitive protective reflex is stimulated, such as "auditory jump reflex," although the efferent tract necessary for voluntary movement is absent.

This probably explains why so many of the vestibular pathways have not been isolated, there being in fact no one definite bundle of fibres in many instances. In such a case all we are able to do is to study and attempt to interpret an absence or perversities of the whole or part of a normal reflex; and even in gross surgical lesions, in which large areas of cerebral tissue are affected, these perversities may not be present because of the establishment of compensation or because of the absence of definite cerebral localization of the function itself.

It is doubtful whether in very primitive reflex, any definite nerve tissue arc or pathway exists in the cerebral tissue, for we know that in spinal reflexes there is a "choice of efferent paths by which the excitation can be transmitted to the peripheral motor organ."*

Clinical Method of Interpretation Necessary:

If the vestibular tests are to occupy a place of practical value in the differential diagnosis of surgical and non-surgical lesions, I believe it is necessary that the present method of their interpretation from theoretical generalizations, based on supposed

*Luciana: Human Physiology, Vol. III., p. 93.

"interference with this or that" so-called "vestibular pathways", must be largely abandoned, and the abnormal vestibular reactions listed as they are clinically present in different diseases and lesions. It is only by some such method that definite information of clinical value can be obtained from the apparently confusing reactions.

In the present imperfect state of our knowledge regarding the vestibular reactions, the present method of attempted localizing intepretation based upon interference with vestibular pathways is as senseless as to argue that because "fever is a reaction to an irritant" whenever we have an irritant we must have fever. The presence or absence of fever is but one clinical manifestation of many diseases and lesions, and while of great diagnostic and prognostic value, it is of obscure origin.

If, however, the abnormal vestibular reactions are specially listed under a *clinical classification* (of lesions and diseases) as clinical symptoms (of such diseases and lesions); and data in a large number of cases obtained, will then, and only then, become possible to argue backward and to attempt cerebral localization by the assistance of the known vestibular manifestations. It is only by such a slow, laborious, and uninspiring method that real progress is possible.*

Clinical Classifications Under Which Abnormal Vestibular Reactions should be Enumerated and Discussed as Symptoms of:

(A) *Non-surgical Lesions, which because of their more or less* microscopic size are *not associated with increased intracranial pressure:*

(a) Chronic degenerative lesions; syringomyelia, disseminated sclerosis, locomotor ataxia, Friedreich's ataxia;

(b) Vascular disease; minute hemorrhages; small vascular occlusions;

(c) So-called functional nervous diseases, such as chorea, epilepsy, etc.

(B) *Surgical Lesions:* chief among which, because of their constant presence in all surgical lesions, should be enumerated;

(1) *The vestibular manifestitations of increased intracranial pressure in general:*

Having recognized that increased intracranial pressure, especially of the posterior fossa, gives rise to a distinct vestibular manifestation (which should be classed among the symptoms of increased intracranial pressure the same as papil-

*Eagleton, W. P.: Brain Abscess—Its Surgical Pathology and Operative Technic. Macmillan, 1922. (In press).

loedema), we can then further sub-divide the vestibular manifestations of surgical disease according to the origin and location.

(2) *Localized suppurative diseases of the posterior fossa;*

(a) Protective meningitis from labyrinth suppuration in intrapiarachnoid abscess;

(b) Intracerebellar abscess confined to the hemispheres of the cerebellum;

(c) Intracerebellar abscess invading the vermis, while the vestibular manifestations of:

(3) *Cerebral Abscess should be divided with those present into:*

(a) temporosphenoidal lobe abscess;

(b) frontal lobe abscess;

(c) occipital lobe abscess.

(4) *Surgical Non-suppurative Lesions* with increase of the intracranial pressure, do not lend themselves to analysis with the same facility as localized suppurative diseases, not only because they have not the same tendency that a brain abscess has for the selection of definite sites at the beginning, except in a few particulars, but also, *because* of the *chronic nature of their invasion,* they are not apt to give the clean-cut picture of vestibular manifestations induced by a localized area of acute suppuration; for while tumors (encapsulated) are frequently more circumscribed than brain abscess, compensation has had greater facility for re-establishment. Consequently, it is essential that they be listed separately from suppurative diseases.

The vestibular manifestations of brain tumors should be listed under those present as follows:

(A) *Tumors below the Tentorium:*

(a) Tumors of the acoustic nerve,

(b) Tumors of the pontine-cerebellar angle,

(c) Intercerebellar tumors of the cerebellar hemispheres,

(d) Tumors involving the vermis.

(B) *Tumors of the Cerebrum:*

(a) Pituitary growths,

(b) Suprasellar growths,

(c) Pineal tumors,

(d) Frontal lobe tumors, Anterior to the Rholandic fissue,

(e) Temporosphenoidal lobe tumors,

(f) Motor-tract tumors, Post Rholandic,

(g) Occipital lobe tumors.

I am perfectly aware of the many weaknesses of such a classification, but as it is based upon the usual lesions and their sites most frequently selected, it has some practical value, especially as during the period of invasion and before the establishment of compensation the vestibular manifestations are apt to be the most clear cut, and, consequently, of the greatest assistance.

Accidents During Performance of Vestibular Tests:

In addition to the possible danger of causing a recurrence of a previous purulent otitis by the application of a cold caloric, it should not be denied that the rotation test may, under certain conditions, precipitate alarming or even dangerous disturbances. This has occurred to me in two instances.

Case Reports

Case I: A girl of twelve years, prior to the evacuation of a temporosphenoidal lobe abscess, had a convulsion. The operation was followed by a small hernia which soon became completely covered with skin, and the patient was discharged as cured. Three years later she was recalled for examination, at which time there was a slight bulging about the size of half a marble over the site of the old hernia. The girl apparently had been in good health for three years. In the midst of the rotation she had a convulsion, became unconscious and was apparently dying. From this state she gradually recovered and was able to be returned to her home (about two hours later). When it is recalled that for three years she had had no cerebral symptoms, and that the only previous convulsion undoubtedly was due to her brain abscess, the inference is forced upon us that the rotation caused a severe disturbance, probably vascular, of her brain—made possible, perhaps, by the dural defect.

Case II: A woman, in her fifties, with a high blood pressure, developed a slight exophthalmos, which appeared first in one eye and then slightly in the other. At the time of the examination the veins of both ocular conjunctiva, especially of the left, were distended, as were the veins in both fundi, in which there were multiple small hemorrhages. R. V. 20/50; L. V. 20/70. There was a paralysis of the left external rectus. A distinct bruit could be heard all over her head, chiefly on the left side, which she said had been present off and on for the previous eighteen months, coming on suddenly and then gradually subsiding. (This and the diminished hearing in both ears, but especially in the right —the contralateral side—had been attributed to her high blood pressure.) The Weber was referred to the right. She gave a history of severe headache, accompanied by vomiting and swelling of her lids.

A diagnosis of pulsating exophthalmos from aneurismal varix of the internal carotid, with rupture into the cavernous sinus of the left side, was made. During her neurological examination it was discovered that she had a *spontaneous deviation* of the arm of *the contralateral side inward*, about two inches. I ordered that she be rotated. Rotation to the right; horizontal nystagmus, 24 seconds; no vertigo, but she vomited; rotation to the left; horizontal nystagmus, 23 seconds; no vertigo. Immediately following the rotation she had a severe pain in her head and began to vomit; both eyes protruded greatly, associated with such an intense oedema of the skin that the eyes could not be opened, and there were numerous fresh hemorrhages in both retina.

On the following day she was able to recognize hand movements in one eye, the other being totally blind except to light.

This attack resembled her previous attacks, but was much exaggerated. For several days I feared she would die. Remarkable to relate, during this time the pulsation in her head entirely disappeared, both subjectively and on auscultation, and never recurred.

Four days after the rotation I ligated the left common carotid artery in the neck. This was promptly followed by a recession of the eyeballs, and later by absorption of the hemorrhages.

One month later, R. V. 20/60; L. V. 6/200. The vision of the right eye —the contralateral—now began to fail; the conjunctival veins enlarged and fresh hemorrhages appeared. The hemorrhages were associated with an attack of glaucoma of the right eye, which rapidly caused her to become blind in that eye, while in the left—the primarily affected eye—the vision improved and today she has a vision in her left eye of 20/30.

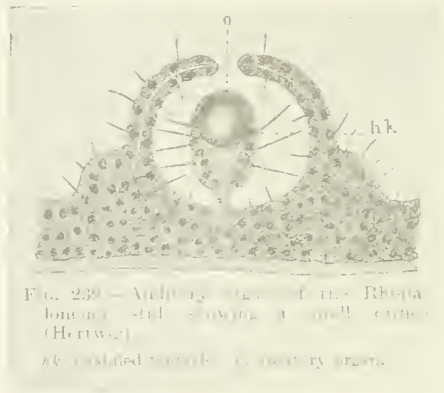


Figure 2. Vestibular Apparatus In Butterfly Showing Highly Developed Vestibular Apparatus But Absence of Auditory Apparatus.
From Morat, J. P.: Physiology of Nervous System. Keener, Chicago, 1906.

As in the previous case, it undoubtedly was an abnormal cerebral vascular system which caused the accident. There is, of course, no excuse for the latter case; she should not have been subjected to rotation nor a caloric, but with the former it shows that in vascular disturbances, either pathological or traumatic, rotation is not without some risk.

Biological Data of Clinical Significance about the Vestibular Apparatus:

Even in very low forms of life a well-developed vestibular apparatus is present, because orientation is necessary. In the invertebrate, life very early evolved a vestibular apparatus out of one of its most primitive attributes—viz., sensation; for "functionally, the vestibular apparatus is a transformation of the tactile apparatus adapted to analysis of pressure."* Fig. 2. It

*Morat, J. P.: Physiology of the Nervous System; 1906, p. 602.

was not until much later, as we have seen, that life, in the vertebrate, envolved an auditory apparatus for the further analysis of the waves of sound.

This fundamental evolutionary fact that the vestibular apparatus is of primitive origin, well developed in the invertebrate, and audition is of much later development, first appearing among the vertebrates, and then very rudimentarily, is of prime importance; for the simpler the form of life, the more easily is it able to dispense with its parts, or rapidly compensate for the loss of or an injury to them. This explains the ability possessed by man of rapidly compensating for, or entirely dispensing with, this rudimentary apparatus. It furnishes the clue to the clinical facts that the loss of or injury to the vestibular apparatus, both peripheral and central, is rapidly and even completely recovered from by the establishment of compensation, while damage to the auditory apparatus is permanent, there being no compensatory machinery to take its place. A man loses the hearing of both ears from suppuration—his vertigo, nystagmus and spontaneous pointing deviations soon disappear, but his hearing never returns.

During the past few years, as the wonders of recent discoveries regarding the vestibular apparatus have dazzled our intellect and stimulated our imagination, we have regarded the vestibular apparatus as highly specialized, on the same plane with speech, which possesses very definite "tracts", and an injury to which produces definite and irreparable manifestations. In this our error has originated. Because the vestibular apparatus is so *new to us*, we have treated it as new to Life, while in reality to Life it is one of the oldest attributes—not "as old as the hills," but much older; for when Life acquired it the waters covered the surface of the earth, and the hills were not yet.

A. Vestibular Tests in Non-surgical Lesions.

In a consideration of vestibular reactions the presentation of a clear-cut picture—a loss of such parts of otherwise normal vestibular reactions sufficient to warrant a vestibular peduncular symptom-complex or a vestibular posterior longitudinal symptom-complex—must presuppose a very small lesion, so small, in fact, as to be almost microscopic. In multiple sclerosis, syringomyelia and Friedreich's ataxia the vestibular reactions are of the greatest localizing value because the lesions are degenerative and microscopic in extent, without a compli-

cating increase of intracranial pressure. Only in such lesions will the rules formulated for "interference with such and such a vestibular pathway" find a proper application.

B. Vestibular Tests in Surgical Cranial Lesions.

Visualization of the Lesion—not "Interference with a Vestibular Pathway"—Necessary for Surgical Localization:

In considering the vestibular reactions in their relation to surgical lesions of the brain, the surgeon should not regard the intracranial portion of the vestibular as definite inflexible tracts, which are either opened or closed as an electric circuit. Unfortunately, surgical cerebral pathology is not so simple.

The surgeon must think of something which can be definitely visualized—an isolated area, such as an endothelioma, cyst, or extradural hemorrhage, or a portion of the brain surrounded by a zone of induration, such as an abscess or an infiltrating glioma.

All surgical lesions cause an increase in the intracranial contents, and give rise to profound disturbances in all parts of the intradural tissues, influencing the vestibular reactions along with the rest, although the growth or abscess may not be situated directly in the course of any vestibular neuron. Consequently, when we attempt to apply the rules of vestibular tract localization, (which from a pathological standpoint may be accurate), the surgeons must not leave out of consideration the vestibular manifestations of the increased intracranial pressure itself, which not only modify but frequently overshadows the other vestibular manifestations.

Factors Influencing and Altering the Vestibular Reactions in Surgical Lesions:

I. Increased intracranial pressure,

II. Establishment of compensation in the two vestibular apparati,

Corollary Factors:

III. Duration of the lesion—whether acute or chronic,

IV. Nature of the lesion—whether causing symptoms (a) by pressure alone, as in endothelioma, cysts, intradural or intrapial hemorrhages, or (b) by destruction or infiltration, abscess or infiltrating tumors.

(a) The Vestibular Manipulations of Increased Intracranial Pressure.

Through ignorance of the work of other observers, the vestibular manifestations of increased intracranial pressure have been largely disregarded. *The vestibular manifestations of increased pressure, especially of the posterior fossa, are definite,* and so outspoken as to overshadow, and in the majority of cases, to prevent the localization of surgical lesions by the vestibular tests alone, even when an intracranial portion of the vestibular apparatus itself is directly involved, as in many cases of large cerebellar abscess; for *in surgical lesions* it must be realized that the *vestibular manifestations which stand out* most prominently are *the manifestations of increased intracranial pressure,* and these vestibular manifestations are disclosed only by the caloric tests, just as the ocular manifestation of increased intracranial pressure—papilloedema—is disclosed only by the ophthalmoscope.

A large personal experience would lead me to postulate that in the presence of increased intracranial pressure the vestibular reactions are of *great general diagnostic value, although they are but rarely useful in cerebral localization.* In my opinion, they should take, like the demonstration of a papilloedema by the ophthalmoscope, their position as of great clinical assistance in the diagnosis of increased intracranial pressure, especially of the posterior fossa.*

In the year 1912, in a communication to the American Otological Society†, after recording the history of three cases of tumor of the brain involving the posterior fossa, and commenting upon *the absence of reaction to the cold caloric when applied to the ear of the opposite side of the lesion* in all three cases, I stated; " This condition, if generally present, may be of service in *diagnosing increased intracranial pressure* involving the auditory apparatus, possibly at an early stage," and I concluded with the statement: "A study of these three cases leads me to believe that in *intracranial lesions of the posterior fossa there may*

NOTE:—Prior to the publication of this paper I had applied the cold caloric in the upright position, thus stimulating the vertical alone. I had no knowledge that in cases of increased intracranial pressure of the posterior fossa, a vestibular reaction follows when the opposite horizontal canal is stimulated.

*It is strange that this error has been made, because of the former fallacy regarding the localizing importance of papilloedema.

†Eagleton, W. P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin: Report of Three Cases with a Previously Undescribed Aural Condition. Transactions of the American Otological Society, June 10-11, 1912, Vol. XIII, Part III, p. 564. Also, Laryngoscope, 1913, Vol. XXII, p. 592.

occur early reduction of the excitability of the vestibular apparatus, not only of the affected side but of the unaffected side as well.

Prior to the appearance of my paper there was no recorded observation, although Neumann previously had called attention to the reduced irritability to the cold caloric of the contralateral side in suppurative otitis. Consequently, it is claimed that my observation was the first recorded vestibular manifestations of increased intracranial pressure, and more important still, that at that time *the first proper interpretation* was offered.

Since 1912 the accuracy of this vestibular manifestation of increased intracranial pressure has been substantiated in so many instances, both clinically and at post-mortems, that I have come to regard it as of the first importance. In my opinion, it is the failure to appreciate its uniformly overshadowing presence that accounts for many of the disappointments and for much of the scepticism in regard to the value of the reactions in surgical lesions.

Because of its great importance to the clinician credit for whatever value the description and interpretation of the reactions possess is due to this early observation.

The vestibular manifestation of increased intracranial pressure, viz., the absence of reactibility to the cold caloric on the opposite side, is frequently mentioned in the cases reported by other observers, but its diagnostic value apparently has not been appreciated. During the past few years this vestibular manifestation of increased intracranial pressure has been elicited by me in a large number of cases of various intracranial pathological processes. In numerous instances its reliability has been proved by a lumbar puncture; and on several occasions an amber colored fluid under pressure—showing a pathological condition—has been encountered in cases in which the routine otological examination had led me to believe a peripheral lesion was present.

I can confidently affirm that the demonstraation of an increased intracrainial pressure by the caloric has largely revolutionized my conception of many of the poorly understood intracranial lesions, such as cases of deafness, psychoses, and headaches following trauma, and in several instances as well has rendered possible a diagnosis of probable brain tumor long before the appearance of a papilloedema.

Case III. The Vestibular Manifestations of Increased Intracranial Pressure, Diagnosing a Progressive Deafness as of Intracranial Although Apparently of Peripheral Origin; Lumbar Puncture Verifying the Diagnosis.

P. G. Male, 29 years of age.

Outstanding Symptoms:

Increasing deafness in both ears, especially the right, during the past five years; tinnitus in both ears; severe headaches.

Family History in regard to hearing—negative.

History:

No preceding illness. For six years he has had loud noises in his ears, and has been growing deaf gradually in both ears, with headaches and slight dizziness in moving or turning his head. He never had pain in nor discharge from his ears. The conditions seem to grow worse each winter but remain stationary in the summer. He hears better in quiet surroundings and on clear days. The tinnitus in the right ear resembles escaping steam; in the left, a booming sound. The patient believes deafness was noticed before the tinnitus. One year ago his tonsils were removed; within the last year he has had two operations on his nose—all to improve his hearing but without benefit.

Examination: O. E. negative. *Vision* normal with proper correction. *Mouth*: Teeth showed the gums of upper jaw red high up near the fold of the membrane. X-ray of teeth negative. *Nose*: The left inferior turbinate seemed to run far back and hang low posteriorly; membrane glossy; no pus. Transillumination showed no definite shadow but was not bright on either side. Blood pressure 128 over 90.

Hearing: Watch heard at one foot; whisper at five feet; voice across room.

	R.		*Weber.*
A. C.	40	seconds	Head; both ears same; Teeth Left. Chin, Left.
B. C.	110	seconds	
			Rinne.
	L.		Head: Hears same in both ears, makes no difference which ear is plugged. Teeth: Same as head. Chin: Best in Left ear
A. C.	65	seconds	
B. C.	115	seconds	

Vestibular Reactions:

Chart I. P.G.

R Spontaneous

Nystagmus - 0
Past-pointing -
Dizziness - 0
Nausea - 0

Rotating to Right

Nystagmus - → 46 seconds; good amplitude.
Past-pointing - +
Dizziness - +
Nausea - +

Rotating to Left

Nystagmus - ← 44 seconds; good amplitude.
Past-pointing - +
Dizziness - +
Nausea - +

October 11:

R Cold Caloric in Right

Vertical

Nystagmus -
Past-pointing -
Dizziness - ++
Nausea - +

Horizontal

Nystagmus - →
Past-pointing -
Dizziness - +++
Nausea - (vomiting) ++

Cold Caloric in Left

Vertical

Nystagmus - 0
Past-pointing -
Dizziness - +
Nausea - 0

Horizontal

Nystagmus - ← +++
Past-pointing -
Dizziness - ++++
Nausea - ++

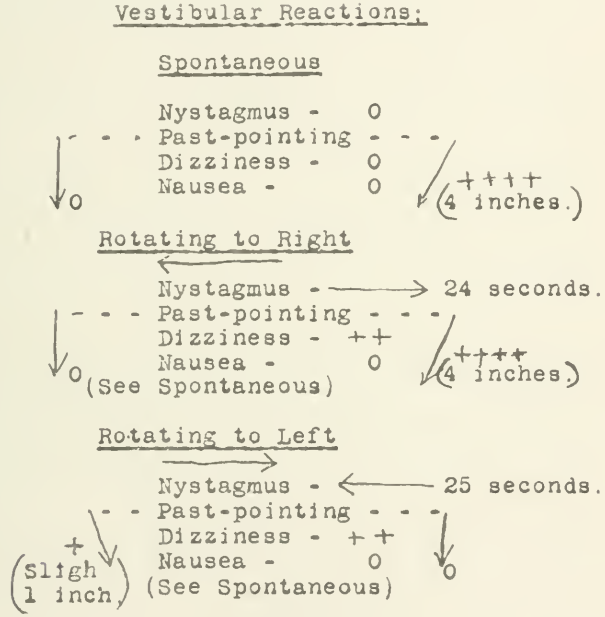
Blood Wassermann Negative. *Lumbar Puncture* gave fluid under *very great pressure, opaque, of a slightly amber color;* globulin and sugar normal; cell count 6.5 per cmm.

Chief Complaint: Progressive loss of hearing, tinnitus with headaches.

Abnormal Findings: Exaggerated patella reflexes; great exaggeration of the duration of the nystagmus; lack of proper pointing deviation; positive Rhomberg. "Left caloric reaction gave no nystagmus, some dizziness, no past-pointing; consequently, the trouble probably is a right-sided lesion."

Diagnosis: "In view of all the symptoms it would seem possible that the man had a disseminated sclerosis. It sounds like a toxic lesion; possibly a thyroid case. The amber-colored cerebrospinal fluid is diagnostic of a degenerative lesion."

January 28:



Increased Intracranial Pressure a Handicap in Localization by Means of the Vestibular Reactions:

Surgeons and clinicians should appreciate that while in a microscopic lesion involving the vestibular tract, exact localization by the vestibular reactions of the site of the lesion, either in the cerebellum, the cerebellar peduncles, the posterior longitudinal bundle, or even in certain parts of the cerebrum, is frequently possible; the presence of increased intracranial pressure of the posterior fossa, causing as it does a uniform absence or reduction of irritability of the vertical canals of the

opposite side, as well as those of the affected side, generally prevents accurate localization by the vestibular tests alone. Much in the same way as an intense papilloedema, by causing a general contraction of the visual field, may obscure a localizing hemianoptic defect (in the visual fields).

In cases of increased intracranial pressure, the reduction of the irritability of the vertical canals of the opposite as well as those of the affected side renders diagnosis impossible by the vestibular manifestation alone.

(b) Establishment of Compensation in the Vestibular Mechanism-Cerebral Reduction of the Duration of the Induced Nystagmus on Turning:

Another factor of fundamental importance, which further interferes with the localizing value of the vestibular reactions and greatly complicates their interpretation in lesions affecting the intracranial portions of the vestibular apparatus, both gross and microscopic, is the early establishment of compensation. To this I also called attention in the paper referred to, as shown in the reduction of the duration of an induced nystagmus and of the caloric excitability of the tracts of the two sides. This readjustment, to which I gave the name of "establishment of compensation", is intimately associated with and allied to the vestibular manifestations of increased intracranial pressure.

It may be imperfectly illustrated as follows: With a normal vestibular apparatus—end-organ and central—stimulation of a horizontal canal by turning produces an induced horizontal nystagmus of 24 seconds' duration, 16 seconds being contributed, it is believed, by the horizontal canal of the labyrinth chiefly stimulated and 8 seconds by the opposite labyrinth.

If one labyrinth is destroyed the maximum duration of induced nystagmus obtainable is 16 seconds, wholly contributed, of course, by the remaining intact labyrinth, while 8 seconds of nystagmus occur on turning in the opposite direction, this 8 seconds also coming from the remaining labyrinth.

In time, however, the duration of the nystagmus is again equalized—although one labyrinth continues out of commission—by, I believe, the establishment of compensation. The duration of the induced nystagmus on maximum stimulation by turning toward the destroyed labyrinth, which immediately after the labyrinthine destruction was 16 seconds, is reduced to 10 or 11 seconds; and the stimulation—on turning away from the lesion—which immediately after the labyrinthine destruction had a duration of 8 seconds, now is increased almost to the same as that recorded in turning in the opposite direction, viz., to 10 or 11 seconds. In other words, a compensation has been established by dividing the duration of the induced nystagmus equally between the two sides, although one end-organ is now contributing all the stimulation.

C. Vestibular Tests in Diffuse Suppurative and Hemorrhagic Diseases of the Meshes of the Piarachnoid of the Posterior Fossa.

Even with these handicaps the vestibular tests, if routinely and conscientiously applied as part of the general neurological examinaton—just as the hearing and sight are tried and the eye grounds inspected—will be found to be of the greatest assistance and will, as our knowledge of the vestibular apparatus is amplified, become of more and more assistance in localization. Progress, however, can be made only by the publication of case reports, so that large numbers of detailed reports may be collected and properly analyzed.*

D. Vestibular Tests in Protective Meningitis of the Posterior Fossa from Labyrinth Disease or Intrapiarachnoid Abscess.

With this in view the following four cases are presented in detail. From them certain deductions are to be drawn. Whether, however, these deductions will be substantiated must depend upon the observations of many independent investigators.

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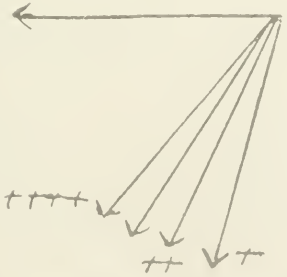
*In the publication of reports of vestibular findings, all details must be incorporated, as the lack of minute details—such as omitting to state the exact position of the patient, whether lying in bed or sitting with the head forward at 30° during the application of the cold caloric—may invalidate its value.

For the past-pointing deviations the following graphic scheme may be used: Each + represents one inch until four ++++, after which the angle should be used.



For accurate recording of details I would advocate following routinely the scheme of examining and recording as suggested in the Guide to a Detailed Neurological Examination in "Fracture of the Skull with Court of Clinical Classification and Guide to Detailed Neurological Examination", Eagleton, W. P., Archives of Surgery, July, 1921, Vol. III, pp. 110-153, in which an attempt is made to compel attention to details both during the examination and when recording them; while for purposes of rapid and visualized comparison, such as induced or spontaneous past-pointing and nystagmus, the author offers the visualizing charts given with the case reports in the present paper.

REPORT OF FOUR CASES OF PROTECTIVE MENINGITIS FOLLOWING LABYRINTH SUPPURATION OR TRAUMA, PROBABLY ASSOCIATED WITH A LOCALIZED COLLECTION OF CEREBROSPINAL FLUID OVER THAT PORTION OF THE ANTERIOR SURFACE OF THE CEREBELLUM WHICH LIES BEHIND THE POSTERIOR SURFACE OF THE PETROUS PYRAMID; WITH A SUGGESTED DIAGNOSTIC VESTIBULAR SYMPTOM COMPLEX—NAMELY, (1) ABSENCE OF COLD CALORIC OF THE VERTICAL CANAL OF THE UNAFFECTED SIDE (FROM INCREASED INTRACRANIAL PRESSURE), AND (2) IMMEDIATE REDUCTION BY 50% OF THE MAXIMUM DURATION OF THE INDUCED NYSTAGMUS ON TURNING, (WHICH WOULD BE OBTAINABLE WERE THE LABYRINTH ALONE AFFECTED; WITH (3) EVIDENCE OF THE INVOLVEMENT OF A PAST-POINTING CENTER IN THE CEREBELLAR CORTEX—AS THE POINTING DEVIATIONS COULD NOT BE ALTERED IN DIRECTION UNDER ANY FORM OF STIMULATION.

Case IV. CASE OF SEROUS MENINGITIS WITH PAPILLOEDEMA, CLOUDY FLUID, HIGH CELL COUNT, FOLLOWING LABYRINTHITIS; AND SINUS THROMBOSIS WITH ABSCESS OF THE JUGULAR VEIN; ASSOCIATED WITH SPONTANEOUS PAST-POINTING DEVIATION OF BOTH ARMS AWAY FROM THE LESION, WHICH COULD NOT BE ALTERED BY STIMULATION, ABSENCE OF OPPOSITE VERTICAL CANAL TO COLD STIMULATION (FROM INCREASED INTRACRANIAL PRESSURE)—AND IMMEDIATE REDUCTION OF DURATION OF NYSTAGMUS TO EIGHT SECONDS.

(Successive charts show the persistence of the pointing deviation until altered by the opposite ear becoming affected, when there was a slight alteration.)

August 19, 1919, J. B., man, twenty years of age.

History: Has always had running left ear with frequent attacks of pain. Always has been subject to headaches. Four weeks previously had pain in the ear—a usual condition. The following day had a severe chill, followed by fever, then a sweat. This was repeated several times during the next four weeks, with considerable headache. Admitted to Newark City Hospital. Examination disclosed chronic mastoid involvement. On opening the mastoid Dr. E. A. Curtis found a large septic clot in the sinus. Condition became very poor during the operation; man thought to be dying. Returned to bed without having ligated the jugular. Did well for a few days, then recurrence of chills and pain. About ten days later I exposed his jugular; found an abscess filling the whole of the interior of the vein itself. *Free pus in the vein.* It was not in the fascia surrounding it but in the vein. On slitting the vein, free pus liberated——no clot. Followed downward and in the lower portion found a clot. Incision in vein went below the level of the clavicle, but did not cause bleeding. Jugular bulb now exposed. Free bleeding obtained from the upper portion of the lateral sinus.

Examination of his eyes prior to the second operation showed no change in the discs. Immediately after the second opration the man complained of dizziness, which continued. Did fairly well, although he lost considerable flesh. Did not look sick enough to warrant any further interference.

Seven weeks after the original involvement, and ten days after the second operation, man had two chills, followed by a period of violent headaches, located in frontal region; headaches would pass away as suddenly as they came. Temperature not much elevated. Examination now revealed double papilloedema. (How long this had been present was not known. It was not present prior to the second operation.) His labyrinth now entirely dead. Had no spontaneous nystagmus, but a *spontaneous pointing deviation of both hands to the right,* (away from the lesion). Cold caloric on left (affected) side produced no nystagmus, no vertigo—dead labyrinth, lost flesh. On walking, would fall to the right side (away from the lesion). Patella reflexes present. Had a slight area of anesthesia—only partial—around the third branch of the tri-facial. Had one severe attack of vomiting. Lumbar puncture revealed fluid cloudy, under pressure. Cell count 160; increased globulin. Repeated blood cultures—all negative. No Romberg.

Deductions from Examination:—"It would seem from the pointing deviation to the right side, falling to the right side—both way from lesion—that the inward pointing center (Bárány's) of the cerebellum opposite to the posterior surface of the petrous portion of the temporal is involved. This is also confirmed by the cerebrospinal fluid, giving a protective meningitis with a slight area of anesthesia. If we rotate him and cannot produce a pointing deviation of the left, this may be confirmed."

Sept. 29, 1919. 1—Rotation to right: No nystagmus; no dizziness; no nausea; no deviation of right arm; slight deviation of left to right. 2—Rotation to left: Nystagmus to right, of eight seconds; very slight dizziness; no nausea; deviation of right arm two inches to left; no deviation of left.

Third Operation: Incision of dura over anterior portion of cerebellum; evacuated considerable amount of turbid cerebrospinal fluid; no pus; exploration of cerebellum negative.

Uneventful recovery followed operation.

Subsequent History: May 13, 1920. Right ear has been discharging for some time; during the past two weeks left ear has also been discharging, and man had aching pain back of the ear. Right ear contains large granulations, occluding the canal. Very deaf. Sudden left facial paralysis a few days ago. At times some blurring of vision. O. E. negative BS 20/20.

Right.	Left.
Watch, three feet	Watch, not heard
Whisper, four feet	Whisper, not heard
Voice, across room	Voice, not heard

Rotating to Left—Has nystagmus to right of 9-10 seconds; no dizziness. No deviation of right; left, deviated to *right.* (Tried twice.) (See spontaneous pointing.)

Rotating to Right—Head forward; did not fall; no dizziness.

Rotating to Left—Head forward, did not fall; no dizziness.

Cold Caloric in Right—*Head Erect;* produced no nystagmus or pass-pointing; no dizziness; no nausea; (increased pressure).

Cold Caloric in Left—Did not produce nystagmus; past-pointing; (Dead Labyrinth) dizziness or nausea; with head erect or back.

May 13, 1920:

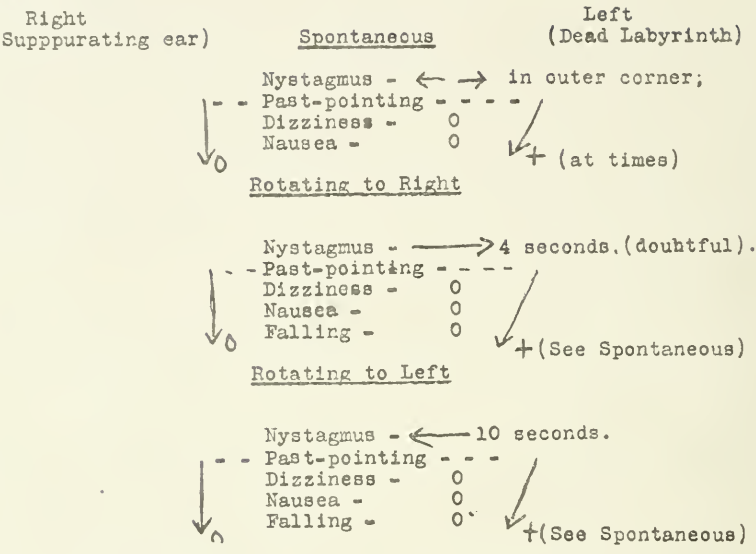
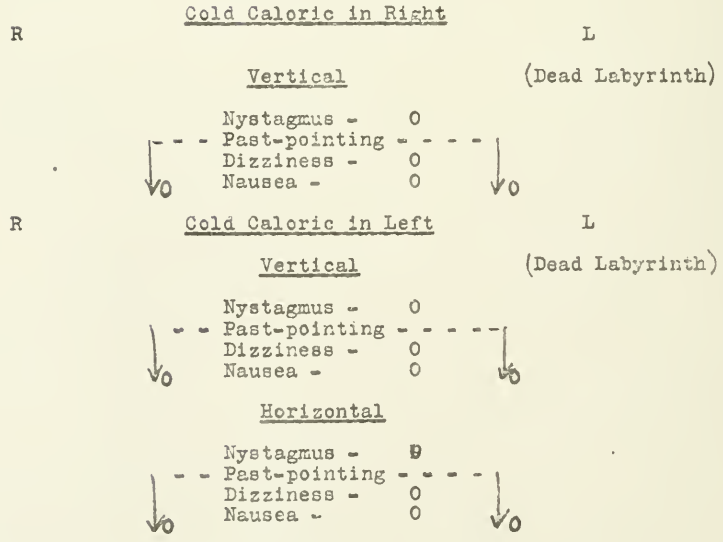


Chart I. J.B.

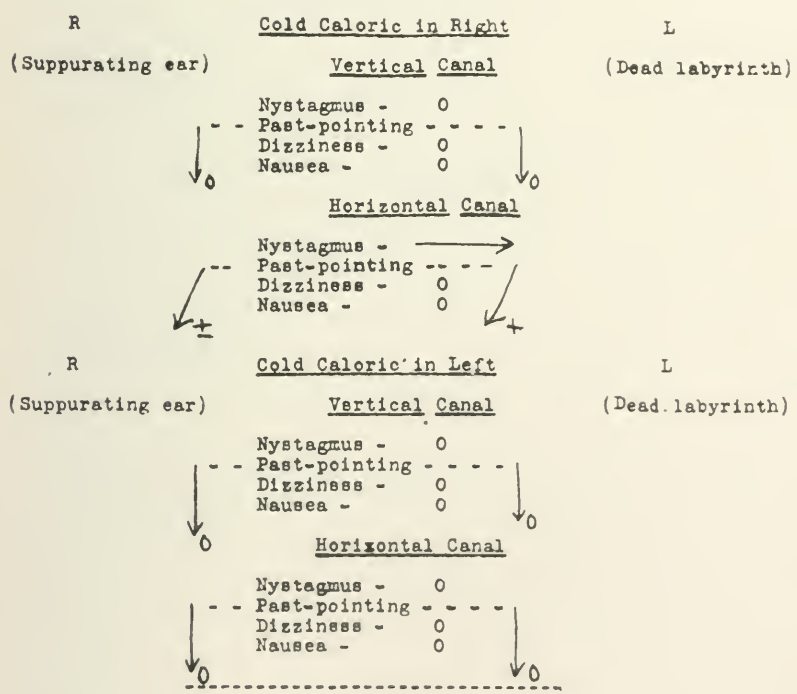


Shortly after the above observations a simple mastoid operation was performed on the right ear.

June 11, 1921. The right ear had continued to discharge from the wound until about three weeks ago, when a large piece of bone came from back of the ear; since then the discharge has ceased. Patient felt well until four days ago; then, sitting down quietly at home, seemingly in usual good condition, he suddenly, on standing up, had severe dizziness, a tendency to fall to the right—which has continued—and nausea at first. The dizziness is not quite so severe now; it came on with a sudden closing of his right ear. The left ear is very deaf following the mastoid on that ear; he has a noise like a faint whistle in the left ear; no pain in ears; dopey feeling in head. Blood pressure 102-68.

Outstanding Symptoms: Sudden closing of the right ear and at once severe dizziness; hearing reduced in that ear. Blurring vision in walking since present dizziness, which clears when he is not in motion.

Chart II. J. B.

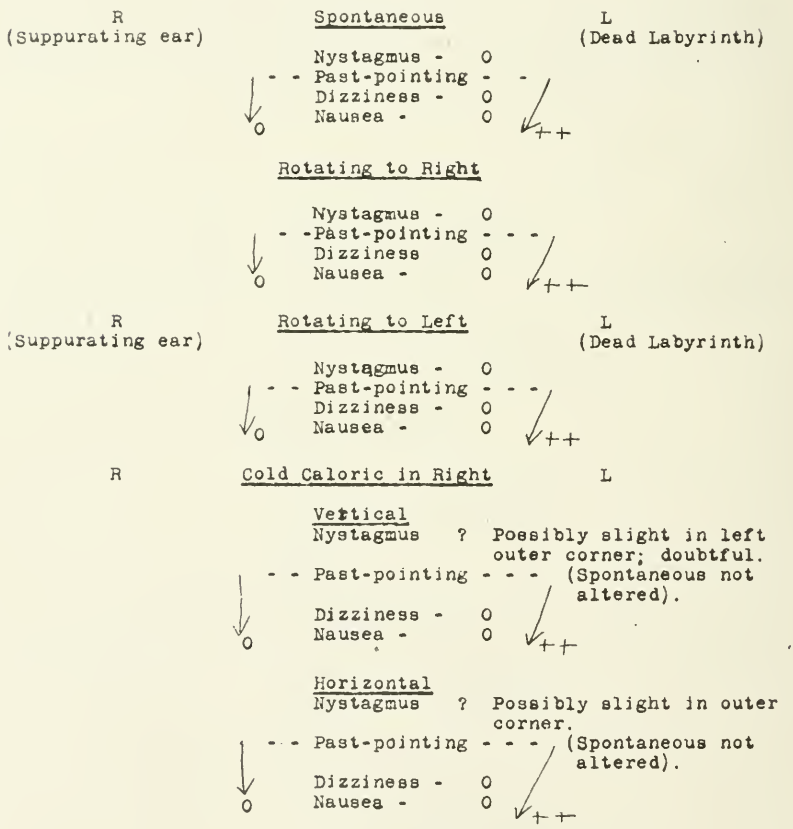


August 19, 1921. Has become totally deaf; is very dizzy and complains of pain over left eye. Sight is reducing. A discharge, with odor, from right ear for two weeks. Falls or staggers at once on attempting to rise, from dizziness and in falling *goes to left side.*

Outstanding Symptoms. Lately totally deaf; discharge with odor from right ear; severe dizziness; general weakness; pain over left eye.

Right Ear. Hears with noise box in left ear, but words must be shouted. Slight discharge. Granulations at site of drum.

Chart III. J. B.



Case V. Case of Acute Suppurative Labyrinthitis Followed by Serous Meningitis Associated with Vertical Nystagmus; Paralysis of Superior Oblique of Homolateral Side; Slight Facial Paralysis and Deafness of Homolateral Side; Homolateral Arm Slightly Incoordinate; Spon-

TANEOUS POINTING DEVIATION OF BOTH ARMS TO SIDE OF LESION; FLUID CLEAR—UNDER SLIGHT PRESSURE—BUT ELEVATED CELL COUNT; REDUCTION OF DURATION OF NYSTAGMUS ON TURNING.

B. R., History: Earache in left ear following an attack of tonsillitis early in April. Two days after earache the ear opened voluntarily and discharged more or less continually for two weeks.

April 16. An aural consultant diagnosed slight mastoid involvement. Paracentesis performed. April 28: the ear stopped discharging; vomiting and other "gastro-intestinal" symptoms followed. Double vision appeared, one object being seen about four inches lower than the other. Another consultant diagnosed "gastro-intestinal trouble." After bowel movement patient improved. This improvement lasted until following evening, May 1st, when another attack of pain in stomach occurred, and headache; followed by period of improvement until May 4, when there was recurrence of headache and "sick stomach" which again gradually subsided.

May 9. Became languid, vomited, and complained of severe headache. Diagnosis of acidosis.

May 10. Patient remained languid; became sleepy, and vomited twice. Another consultant—Dr. Eugene Murray called. Diagnosis, meningitis.

May 11. Patient admitted to hospital. Complained of constant temporal and occipital headache, with pain in neck; deafness of left ear for past three weeks, with tinnitis and dizziness. (There had been no chill and temperature was but slightly elevated.)

Examination. "Nystagmus, vertical, especially upon looking to the left. Possible paralysis of external rectus of left eye; left eye lower than right, left pupil larger than right. Total deafness of left ear; left drum normal, no tenderness over mastoid. Slight left-sided facial paralysis." Has lost flesh. Tongue protrudes in median line. No difficulty in swallowing.

Vestibular Manifestations. Vertical nystagmus; spontaneous pointing deviation of both arms to left; left arm co-ordinates imperfectly.

Lumbar Puncture. Spinal fluid clear, under very slight pressure; sugar reduction normal; globulin slightly increased; cell count 60 per cubic mm. No organisms.

Pre-operative Diagnosis. Labyrinthitis, with cerebellar involvement. Abscess?

Operation. Drainage of diseased deep cells of petrous pyramid; opening of semicircular canals and cisterna of internal auditory meatus. Incision of dura over cerebellum.

Operation Notes. The mastoid was opened and found full of granulation tissues; no pus; the solid angle between sinus and petrous bone opened; small, deep cells found involved and filled with granulation tissue but no pus; proceeded inward behind posterior and semicircular canals. No bleeding. Was compelled, because of forward-lying sinus, to chisel away posterior arm of posterior semicircular canal, which exposed both canals well; canals contained no pus and no fluid. Pin-point opening found in dura (which may have been operative). Discharge of cerebrospinal fluid on reaching internal auditory meatus. Dura over this area covered with granulations.

At completion dura was very black; two or three openings in dura from which cerebrospinal fluid was flowing, but not in great quantity. Middle fossa not exposed; sinus not injured; middle ear was not opened.

Operative Comment. "It would seem that this child has a serous labyrinthitis—possibly a brain abscess *over* the cerebellar region. It might have been better if the labyrinth had been opened through the middle ear and the cochlea exposed as well as the semicircular canals. Exploration into cerebellum was not attempted, although the spontaneous deviation—the lack of good coordination of the same side—pointed to an involvement of the brain stem."

At completion of operation the child apparently could not move its arm on that side, but this rapidly disappeared. O. E. was negative.

May 12. Temperature 100-99°; pulse very fast, 118-148, and at times irregular.

May 13. (Second day after operation). Pulse still rapid, 130; temperature 99-101°. Patient lies on right side, in which position eyes are deviated toward the right and downward; ophthalmoscopic examination negative, but may be slightly indistinct. Complains of soreness of throat. Slight facial paralysis, about the same as before operation. Left arm moves well, but incoordinately, Leucocyte count 19,200. Polynuclears, 85%; large Lymphocytes, 9%; small Lymphocytes, 5%.

May 16. Nurse reports "She seems to look at you with the right eye." Left pupil larger than the right. O. E. negative, but both discs whitish. Paralysis of left external rectus. Patient has tendency to look downward and to the right, in which position of eyes there is no nystagmus; there likewise is no nystagmus when looking downward and to the left, but rotary nystagmus on looking upward when patient is in recumbent position. Tongue protrudes in median line; palate goes up on right side; patient complains that when she swallows the solids and liquids "do not go down right". There seems to be a shoulder girdle in which the inco-ordination is most marked when she has shoulder and elbow free. Both vocal cords move normally.

May 18. O. E. negative; temperature normal; pulse still fast—118-130.

May 19. Smear and culture from left ear; streptococci and cocci probably of sarcina group.

May 29. Left pupil larger than right; left eye turns in on looking to right; rotary nystagmus to right, but absent on looking to left when the eye is still (this occurs when either eye is covered). Complete paralysis of the left external rectus. Incomplete facial paralysis. Marked rotary nystagmus in looking upward; left eye slightly lower than right. No dizziness when patient tries to sit up, but in returning to recumbent position patient becomes dizzy.

June 7. O. E. negative. The left disc is whiter than the right, but it may be that the right is too red. Incoordination of the left arm. Left complete facial paralysis; cannot close eye; complete paralysis of external rectus; on closing left eye moves right eye inward without nystagmus, on turning it outward to the left there is nystagmus. With both eyes open, on looking upward there is rotary nystagmus of vertical tendency; no nystagmus on moving to left, but of course the left eye does not go outward because of paralysis of left external rectus; left eye is downward, and on moving, raises the upper lid; looking to right, no nystagmus. No nystagmus on looking downward, but marked vertical nystagmus on looking upward. Apparently has a paralysis of the superior rectus or of the superior oblique—I think of the superior rectus, but I am not sure. Temperature, normal; Leucocyte count, 9,000; Polynuclears, 67%; large lymphocytes, 7%; small lymphocytes, 25%; eosinophiles 1%.

June 8. *Vestibular Reactions Spontaneous.* "No spontaneous past-pointing; spontaneous rotary nystagmus to right at short intervals; the nystagmus is more noticeable when the patient looks to the right and somewhat upward. It is more constant in this position than when she looks straight ahead; it is also more marked when she looks upward. When she looks straight ahead the act of blinking seems to increase the nystagmus."

Rotating to Right. (1) Right hand deviated two inches to right—did not deviate more than once. Left hand did not deviate. Dizziness; no nausea. (2) No deviation of either hand. No perceptible nystagmus was induced.

Rotating to Left. No deviation of either hand; horizontal nystagmus to right of 8-10 seconds. Dizziness. No nausea.

Cold Caloric in Left. Head erect; produced no reaction. (Dead labyrinth). Head back; no perceptible nystagmus induced, tho possibly slightly horizontal to left. Dizziness. No past-pointing.

August 14. Double vision improving. Images 6 inches; at times vision is single. Image separates on looking to the left. The child apparently is perfectly well. Wound healed.

Case VI. Case of Generalized Protective Meningitis, Result of Suppurative Labyrinthitis, with Vestibular Manifestations of Increased Intracranial Pressure—Non-Reactibility of Opposite Vertical Canal to Cold Caloric—From Which a Diagnosis of Increased Intracranial Pressure Was Made; Reduction of Duration of Nystagmus on Turning; Inability to Cause Arms to Deviate Toward Side of Lesion; Lumbar Puncture Revealed Pressure and High Cell Count—Following Operation Severe Symptoms of Meningitis.

J. G.: male; adult. Seen with Dr. Baldwin November 24, 1919.

History. Had discharging right ear for three months. For the last three weeks had pain in right ear with severe pain in head, in right temporal region; attacks of dizziness during past three weeks—"will be going along and suddenly something occurs and I sway. I have never fallen but have run into things." Does not know to which side he sways. Several chilly sensations and two attacks of vomiting. Last two days facial paralysis.

Examination. Right drum membrane destroyed; granulations and pus in external canal. No hearing in right ear.

Rotating to Right. Head forward; on straightening up, fell to right; dizzi- No dizziness or nausea. Wide deviation of right to right (10 inches). Slight deviation of left to right (3 inches).

Rotating to Left. Horizontal nystagmus to right of *five* seconds. No dizziness or nausea. No deviation of either hand; quick movement and large amplitude; wide pointing deviation of both hands to left; dizziness.

Falling.

Rotating to Right. Head forward; on straightening up, fell to right; dizziness and nausea.

Rotating to Left. Head forward. Did not fall; very slight dizziness.

Cold Caloric in Right. Dead labyrinth.

Cold Caloric in Left. Produced no nystagmus—if any it was horizontal to left—no pointing deviation of either hand, no dizziness or nausea.

Throwing Head Back. Produced a violent horizontal nystagmus to right. Temperature 99°. Pulse 76.

November 27. First lumbar puncture performed; fluid clear, under pressure. Globulin increased. Fehling's slightly reduced. Cell count 100 cm. No organisms.

November 28. Second lumbar puncture; fluid clear, under pressure; globulin increased; sugar reduction not normal; no organisms.

November 29. Operation. Labyrinth opened; fistula found; granulations. Neumann's operation. Posterior fossa opened behind posterior pyramid; a quantity of cloudy cerebrospinal fluid evacuated.

Following operation patient had severe pain over left eye, numbness of fingers of left hand; was restless at night; temperature 101°-103°; Leucocytes count, 12,800; Polynuclears, 73%.

December 3. Stiff neck; severe pain in head; Leucocyte count, 16,400; Polynuclears, 85%; smear from wound, staphylococcus, streptococcus mucosus capsulatus. Temperature 102°-104°; pulse slow 82-90.

From December 3 to December 28 patient continued with restlessness at night, slightly delirious with alternating attacks of stupor, from which he could easily be aroused; severe pain in head; stiffness of neck; Kernig; pain behind left eye; photophobia, with a continuously high temperature—101°-105° —and a proportionally slow pulse. Several blood cultures were negative. Leucocytosis.

December 26. Third lumbar puncture; no pressure; color pale yellow; globulin greatly increased; sugar reduction, about one-half normal; cell count, 633 cm.

January 7, 1920. Patient looked very ill; pulse very fast and of poor quality; but rested better at night; still complained of stiffness in back and legs, but head felt better.

January 18. Patient much better in every way; temperature normal.

February 24. Patient discharged from hospital feeling fairly well but with a discharging ear. A few days later he discharged a sequestra of cochlea, after which the ear promptly healed and all symptoms disappeared.

A similar case with dead labyrinth, high temperature, delirium, almost maniacal headache, double papilloedema, cloudy spinal fluid, with cell count of over 3000 but without microörganisms, an exenteration of the labyrinth, with incision of the dura, was promptly followed by recovery.

Case VII. Result of Cleaning Out Deep Cells of Petrous Developed Labyrinthitis and Serous Meningitis with High Cell Count of Lumbar Puncture Fluid; Spontaneous Rotary Nystagmus and Inward Pointing Deviations of Both Arms, The Direction of Which Could Not Be Altered. (1) Absence of reaction to cold caloric of opposite vertical canal (from increased intracranial pressure) and (2) reduction of duration of induced nystagmus to fifteen seconds giving rise to the supposition that the functional test ultimately would return to normal.

Boy, G. C., seventeen years of age. History. February 22. Scarlet fever, and following this, about February 28 left side, earache with discharge, during which time patient had delirium, and at least one chill. During discharge, had headache over temple. Followed by period of improvement but had discharging ear.

April 12. Mastoid operation by Dr. Leifeld, sinus and dura exposed; apparently improved; no temperature, no headache, until April 21, when patient had slight rigor (possible from ice cream). Next day *pain behind left eye*, which continued off and on. Discharge from ear profuse; no dizziness; temperature 100.2°-102° nauseated and vomiting.

April 26. Lumbar puncture; fluid clear, not under pressure; globulin slighly increased; cell count, 5; no organisms; blood culture, negative; O. E. veins full, no indication of injection of disc. Hearing greatly reduced but present in left ear.

April 27. *Second Operation.* Eradication of deep cells of petrous. Decided not to open his dura. At completion of the operation I was not entirely satisfied; "the boy probably had a serous meningitis, although the low cell count—only three—would signify otherwise."

Granulation tissue found in the mastoid wound. The deep mastoid cells behind the external semicircular canals also opened. There was a large amount of carious bone along the zygomatic process where the dura was freely exposed. The sinus was covered by a thick granulation. Made a small opening in the sinus; had bleeding, but not very free.

April 28. Vomited. Had some dizziness following the operation. "He does not look entirely right." He had a white cell count of 17,000. Blood culture negative." I am still of the opinion that he has an involvement of the temporosphenoidal lobe."

April 29. "Since the operation the boy has been slightly dizzy, and on changing his position the dizziness is increased." Has a nystagmus on looking toward the right(away from the lesion) "It is possible that during the operation there was some injury done to his labyrinth, although I think not." Today appears slightly dopey, but his mother says he always sleeps considerably when sick. Temperature 101°. "I am afraid he is developing serous meningitis. It may be this is due to the injury of his labyrinth, because the deep cells of the apex of the petrous portion of the temple undoubtedly were involved."

Second Lumbar Puncture. Fluid under great pressure; macroscopically clear; globulin slightly increased. Sugar, normal. Cell count 322.5 per cubic mm. Smear shows no organisms. Culture sterile after 24 hours. Differential, polynuclears 93%, lymphocytes 7%.

April 30. Temperature irregular—90.2°-102°. Appears much better; is not dizzy. Total deafness of left ear.

Vestibular Reactions Spontaneous; rotary nystagmus to right on looking straight ahead; more noticeable on looking in right outer corner. *Spontaneous* deviation of *right hand two inches to left.* Tendency for left hand to deviate to *right.*

Rotating to Right. No perceptible change in the spontaneous rotary nystagmus; (possibly stopped for three or four seconds). No dizziness or nausea. Right hand deviated to left (see Spontaneous Deviation). Left hand deviated to right (see Spontaneous Deviation).

Rotating to Left. Spontaneous rotary nystagmus markedly increased for about 15 seconds; then about the same amplitude as before rotation. Dizziness. No nausea. Right hand deviated to left (no increase over spontaneous deviation). Left hand deviated to right (see Spontaneous Deviation).

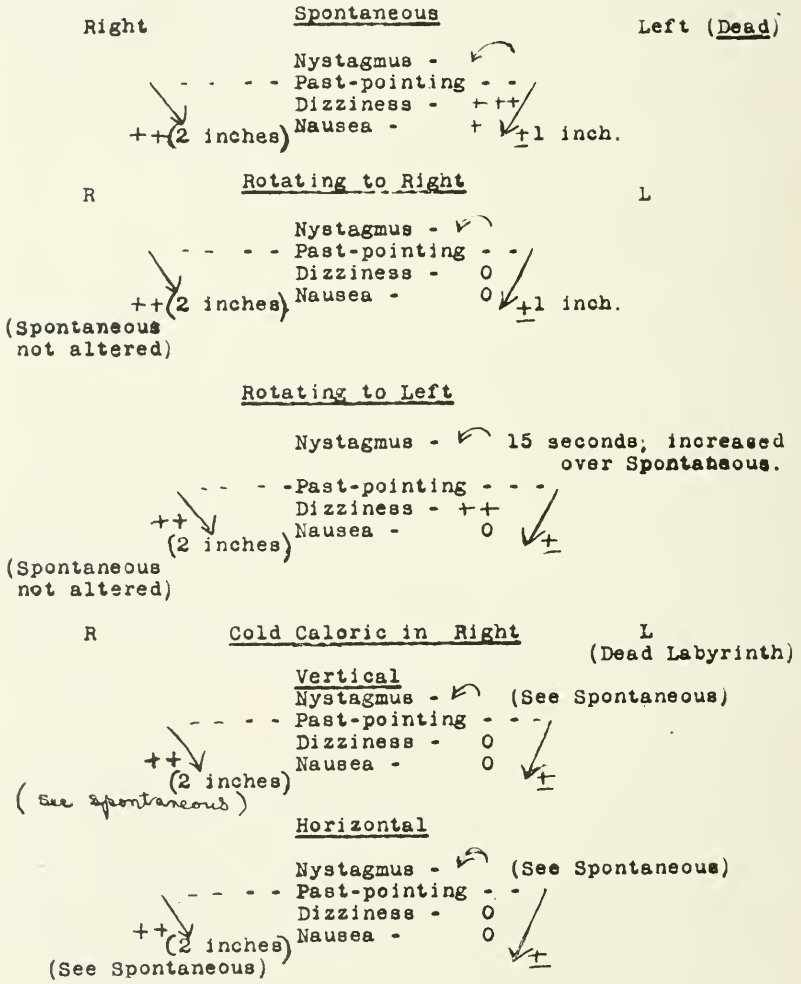
Cold Caloric in Right:

Head Erect: The spontaneous nystagmus to right was not altered; no change in spontaneous deviation; right deviated to left—left deviated to right; no dizziness; no nausea.

Head Back: No perceptible change in spontaneous nystagmus; right hand did not deviate (see Spontaneous Deviation); left hand deviated to right.

Chart I. G. C.

Graphic Representation of Above April 30:



May 2. Patient much better in every way, but still has irregular temperature—102°-102.2°. No dizziness. Leucocyte count, 13,400; differential, polynuclears, 78%; L. lymphocytes, 10%; S. lymphocytes, 11%; eosinophile, 1%.

Blood culture taken; negative. Spinal fluid; slight opacity of fluid, considerable pressure; globulin slightly increased. Sugar, normal. Cell count, 842 per cubic mm. No organisms in smear. Culture. Differential, polynuclears, 95%; lymphocytes, 5%.

May 5. Spinal fluid under considerable pressure; fluid opalescent and showed slight flocculent precipitate. Globulin slightly increased. Sugar normal. Cell count 600 per cubic mm. No organisms in smear. Fluid cultured; no organisms. Differential, polynuclears, 90%; lymphocytes, 10%.

May 6. Leucocyte count 8,800. Differential, polynuclears 72%; L. lymphocytes, 13%; S. lymphocytes, 14%; eosinophile, 1%. "This is the third lumbar puncture which has been done. At the first one before the operation for his labyrinth he did not have a high cell count. A short time after he had a cell count of 150. Three days later it rose to over 300. It is now over 600. The boy, however, looks perfectly well. He is pale. He has no other symptoms. In view of the fact that none of his labyrinthine symptoms came on until after the operation, and we know that he heard before that, and that his nystagmus developed the following day, and that he is dizzy, we had decided that it must be a protective process. We are impressed with this idea because of our experience with case J. G., Case VI, who after a labyrinthine operation, continued to have a high cell count with some rigidity of the neck. However, one month after the operation, he still had a cell count of 600. Some weeks later a small piece of sequestra from the labyrinth came away, and the man's condition immediately cleared. This makes us think that in a chronic case there is a protective meningitis going on along the posterior surface of the petrous portion of the temple. In opposition to this, however, is the fact that he had before the operation a severe pain behind his eye, that he had one chill, and was delirious. The diagnosis of the case must remain between three things; one, of protective meningitis, the result of the labyrinthine operation and the development of a protective area so as to cut off the ductus endolymphaticus, or the internal auditory meatus tract; second, an encapsulated abscess of the temporosphenoidal lobe, now at least of from four to six weeks' duration; or third an encapsulated serous meningitis, either along the posterior portion of the petrous or over the tegmen."

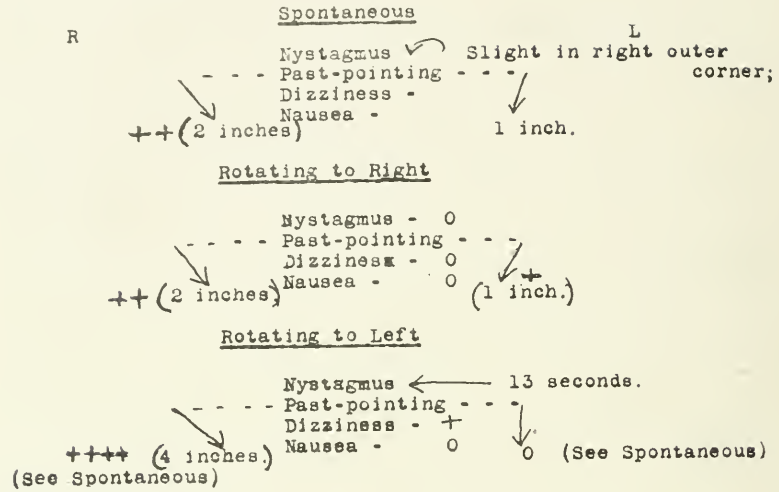
Vestibular Reactions:

Head Erect: Spontaneous, slight nystagmus with rotary tendency to right in right outer corner. Spontaneous deviation of right, two inches to left.

Rotating to Right. No perceptible nystagmus; no dizziness or nausea. Left hand deviated to right; right hand deviated to left. (See Spontaneous Deviation).

Rotating to Left. Has nystagmus to right, of good amplitude, lasting 13 seconds. Slight dizziness; no nausea. Right hand deviated four inches to left; left hand did not deviate. (See Spontaneous Deviation).

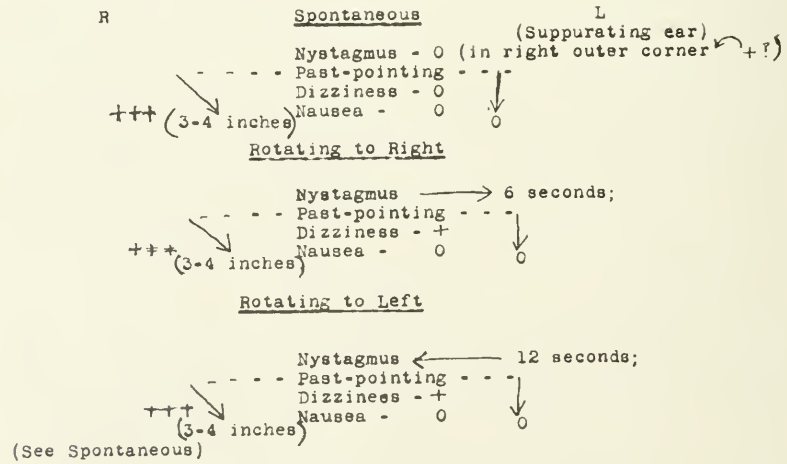
Graphic Representation of the Above:



May 7, 1921. Totally deaf. Slight nystagmus in looking to right; no adiadokokinesis; no heminanopsia of color on rough test; no pointing deviation; O. E. about same; veins full but not tortuous.

Chart III. G. C.

Wednesday, June 29, 1922:



Hearing:

	Right ear:	Left ear:
Watch -	Six feet	Totally deaf.
Whisper -	Across room	
Voice -	Across room.	

It would seem from studying the foregoing four cases that immediately on the increase of the intracranial pressure in protective meningitis or intrapiarachnoid abscess of the anterior surface of the cerebellum, (1) the nystagmus normally induced from the remaining functioning end-organ is reduced from the normal sixteen seconds to about half that duration—namely, to eight or nine seconds—and (2) the spontaneous past-pointing of the arm caused by the involvement of the cerebellar cortex situated posterior to the bony labyrinth cannot be altered by stimulation. Both of these observations would suggest a localizing diagnosis of the lesion.

A study of these cases furnished me a clue to that larger class of cases now so frequently seen, which, following an injury, such as fracture of the skull with cerebral trauma, or associated with a chronic progressive deafness, or from intoxications of all kinds, syphilis and so on; in fact, any condition which lessens the reactibility of the horizontal fibres to a particular form of stimuli (repeated turnings or repeated aerial flights, the clinical signs of which rapidly disappear)—immediately develop a great reduction in the duration of the nystagmus on turning; showing that while the fibres of the vertical canals apparently are much earlier attacked by an increase of the intracranial pressure when stimulated by the cold caloric, the horizontal fibres also are easily affected by any disturbance, as far at least as stimulation by turning is concerned.

Pointing Deviation:

B (Case IV) had bilateral past-pointing away from the lesion; homolateral—pointed inward; contralateral—pointed outward, with falling away from the lesion.

The spontaneous pointing deviation could not be altered by either turning or caloric. After opposite labyrinth became affected the homolateral arm of the second labyrinth was altered and pointed inward (away from the second lesion toward the first lesion), and again the spontaneous deviation could not be altered. In B the contralateral side apparently lost the capacity for stimulation. It stopped deviating in May, 1920; the contralateral continued reactible for a time.

C (Case VII) had bilateral past-pointing deviation, *inward*, which could not be altered. The contralateral side had a greater pointing deviation than the homolateral side. The homolateral side later could not be made to deviate at all, while the contralateral side still continued inward.

R (Case V). There was no past-pointing deviation, but the records are so few, and the child was so sick that it cannot be accepted as of any great value.

These four cases, in a general way, confirm the opinion that the spontaneous past-pointing deviation cannot be altered until at least the lesion has been removed as in the cases reported by Lowenstein. He, with Barany, thinks there is a spontaneous past-pointing deviation of the homolateral side outward.

The fact that B, C, and R could not have the spontaneous past-pointing deviations altered long after the inflammatory process had subsided shows that in these cases of protective meningitis the cerebellar cortex is affected (by the inflammatory process).

These cases would certainly tend to show that whatever mechanism is involved by turning, in diffuse suppurative disease in the meshes of the piarachnoid over the cerebellar cortex, this mechanism is suddenly impaired to such an extent that it does not recover. The influence of this past-pointing deviation would appear the same for both the spontaneous homolateral and contralateral—the past-pointing deviation could not be altered; and in some of the cases at least the homolateral deviation continued reactible while the contralateral was non-reactive.

II. *The Diagnostic Value of a Reduction of Intracranial Origin in the Duration of the Nystagmus from Turning in Diffuse Lesions of the Posterior Fossa:*

I am persuaded that in the cerebellar cortex of the posterior fossa there is something which has to do with this. It is probably in the cerebellar cortex because cortical lesions are the ones that induce it quickly (as in the cases reported), while in intra-cerebellar lesions such as intra-cerebellar abscess, this reduction of the obtainable nystagmus from turning is not present.

My own experience with this reduction in cases of fracture of the skull of the posterior fossa, leads me to believe that it is the filling of the piarachnoid pathways of the posterior fossa with either exudate (as in the herein reported cases of protective meningitis) or with blood as in linear fractures of the skull, chiefly of the posterior fossa, that gives rise to inflammatory and reactive changes which affect the cerebellar cortex; for the reduction of the duration of the obtainable nystagmus by turning occurs immediately, as proven in a case of meningitis which recovered

after subarachnoid irrigation. The reduction in the deviation of the nystagmus was demonstrated one week after the onset of the disease. When, however, the exudate is chiefly confined to the cerebrum, the reduction of the induced nystagmus from turning either does not occur at all or is not permanent as another case of suppurative meningitis which recovered following cerebral and spinal subarachnoid irrigation, had a normal deviation of the nystagmus a few months later.*

Induced Nystagmus from Turning and from Heat or Cold Physiologically and Diagnostically Different:

From a misconception we have in the past regarded the induced nystagmus from turning and from caloric as identical. This is physiologically and clinically incorrect, as I have herein demonstrated. There is no evidence to substantiate the view that the induced nystagmus from turning and from cold and heat are identical. We have presumed, however, that they were identical, because objectively they are apparently the same; but judging from physiological analogy it is much more likely that they are in certain parts, at least, separate. The form of stimuli are so dissimiliar—turning being a mechanical stimulus which is well adapted to cause a molecular disturbance in a fluid medium (such as the fluid of the labyrinth) analogous to a certain degree to stimulation of the proprioceptive sensory system —deep sensation— while cold and heat are thermic stimuli ill adapted to such disturbance, but, on the other hand, well adapted to stimulation of the nerve fibres by cutaneous sensation. As both are forms of sensory stimuli, the vestibular apparatus being one of the early specialized apparati to convert sensory impulse into voluntary and involuntary muscular contractions, the different sensory impulses from turning and caloric probably are transmitted by the vestibular apparatus in a similar way as the transmission of cutaneous impulses to the cord from pressure or thermal sensations, either by a different set of cutaneous nerves, or at least by nerves differentiated in the posterior roots.

It is possible that the cerebellar cortex *as a whole* has an analogous function to the posterior roots as far as the differentiaton of the vestibular stimulation or impulses is concerned. In turning we are stimulating in a way a muscular sense—the sense of movement, while in caloric we are stimulating a sensory sense—a cutaneous (thermic) sense.

*Eagleton, W. P. Subarachnoid Irrigation for Suppurative Meningitis. Trans. Amer. Otol. Soc., 1921.

Two Cases Illustrative of the Immediate Reduction of the Inducible Nystagmus from Stimulating the Horizontal Semicircular Canals by Turning with Preservation of Normal Hearing in Diffuse Traumatic Lesions of the Posterior Fossa, Such as from Linear Fracture of the Skull.

Case VIII. M. A., girl, 8 years of age.

Resume of Case:

Struck by an automobile; since then has had dizziness and headache, double vision in the distant, not in the near, which may not be pathological but looks abnormal; high degree of hypermetropia with astigmatism; reduced vestibular reaction in the horizontal canal; absence of reaction in vertical canal in left ear; hearing normal.

Impression:

"It would seem then that she undoubtedly had a fracture somewhere through into the posterior fossa. Both vertical and horizontal canals fail to function properly."

History:

In March, 1920, was run down by an automobile and badly injured, especially on the back of her head. She was taken to a drug store and about an hour afterward taken home in an automobile by a policeman. The mother does not know whether or not the child was unconscious at any time. The physician who was called in to attend her, and helped to take her home, attended her for a few weeks at her home. The back of the child's head was much swollen and discolored for more than a month, but the doctor did not regard it as due to a fracture of the skull. The child was in bed for a week after the accident. On getting up she had to be led, but the mother was unable to say whether this was due to dizziness, to general weakness, or to the injury to her right leg.

Patient seemed to recover gradually, but remained very pale, had no appetite, complained of pain beginning at the back of her head and coming over the eyes, and said she was invariably dizzy on first standing up. Since the accident she has been told at school that her sight is not normal. She does not see writing on the blackboard well, and her head aches after reading for a time. When asked if she ever sees double, she replied that "when things are far off they are two." A street car at a distance appears to be two cars, but becomes one as it draws near. The same is true of an automobile; also of persons; approaching at a distance she sees two, but when they are near by she sees them normally.

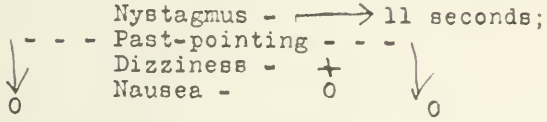
When she becomes excited or worried she feels a soreness or a stiffness in her neck and an aching in her left ear. She did not seem to have any ear symptoms at the time of the accident.

Jan. 11, 1921: Chart 1. M.A.

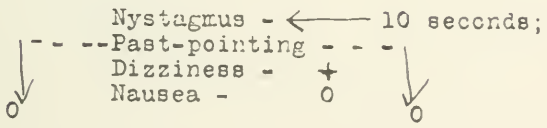
Vestibular Reactions;

No spontaneous nystagmus or past-pointing.

R. Rotating to Right: L

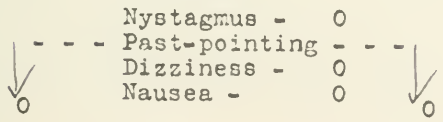


Rotating to Left



R Cold Caloric in Left L

Vertical



Horizontal

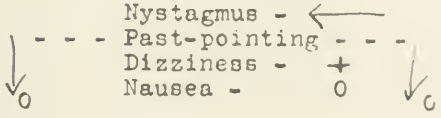


Chart II. M. A.

January 13:

)

R

Cold Caloric in Right

Vertical

Nystagmus - 0

- - - Past-pointing - - -

Dizziness - ?

Nausea - 0

+ + (2 inches) ++ (2 inches)

Horizontal

Nystagmus - 0

- - - Past-pointing - - -

Dizziness - ?

Nausea - 0

+ + (2 inches) ++ (2 inches)

Feb. 16:

Report of Roentgen Examination:

"Plates were made of the head in both lateral positions and antero-posterior position with the occiput on the plate.

"They show a small line of fracture on the right side in the occipital bone just below the occipito-parietal suture. This is seen in both right lateral and antero-posterior views."

Chart III. M. A.

Feb. 18:

Spontaneous

R L

Nystagmus ⟶ Slight, in left outer corner.
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Right

Nystagmus - ⟶ Possibly 7 secs,;slow movement;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Left

Nystagmus ⟵ 7 or 8 secs.; slow movement;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Cold Caloric in Right

R L

Vertical
Nystagmus -
Past-pointing - - -
Dizziness - ++
Nausea - 0

Horizontal
Nystagmus - ⟶
Past-pointing - - -
Dizziness - ++
Nausea - 0

Cold Caloric in Left

Vertical
Nystagmus -
Past-pointing - - -
Dizziness - + ?
Nausea - 0

Horizontal
Nystagmus - ⟵
Past-pointing - - -
Dizziness - +++
Nausea - 0

February 24:

Wasserman - negative.

Chart IV M.A.

Feb. 29: Vestibular Reactions

Spontaneous

R L

Nystagmus ⟶ Slight in left outer corner;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Right

Nystagmus ⟶ Possibly 7 seconds; slow movement;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Left

Nystagmus ⟵ 7 or 8 seconds; slow movement;
Past- pointing - - -
Dizziness - 0
Nausea - 0

Cold Caloric in One Ear - the Right

Vertical

Nystagmus -
Past-pointing - - -
Dizziness - ++
Nausea - 0

Horizontal

Nystagmus ⟶
Past-pointing - - -
Dizziness - ++
Nausea - 0

Case IX: J. N., fireman, 26 years of age.

History: Previous history of good health, with no history of acute illnesses. Fell from third story window when infant of three years. Nov. 24, while driving engine which collided with trolley car, patient was thrown from engine to street. Was admitted to hospital suffering from abrasions, contusions and possible fracture of the skull.

Examination: General appearance of well-nourished, well-developed white male of 26, not acutely ill, lying quietly in bed, conscious and rational.

Head: Scalp, skull and hair, negative.

Eyes: Contusion over left eye, with a sub-conjunctival hemorrhage; pupils equal, regular, react to light, sclera clear in right eye, no nystagmus, no strabismus. Eye-grounds negative. No facial paralysis.

Neck, heart, lungs, abdomen, extremities, skin, all negative; nodes, not palpable.

Reflexes: Knee and ankle, active and equal; no Babinski; no clonus; no Oppenheim; abdominals and cremasteric active and equal. B. P. 140 over 90.

X-ray showed fracture right occipital region; no depression.

Provisional Diagnosis: (1) Contusion of left eye; (2) possible fracture of skull.

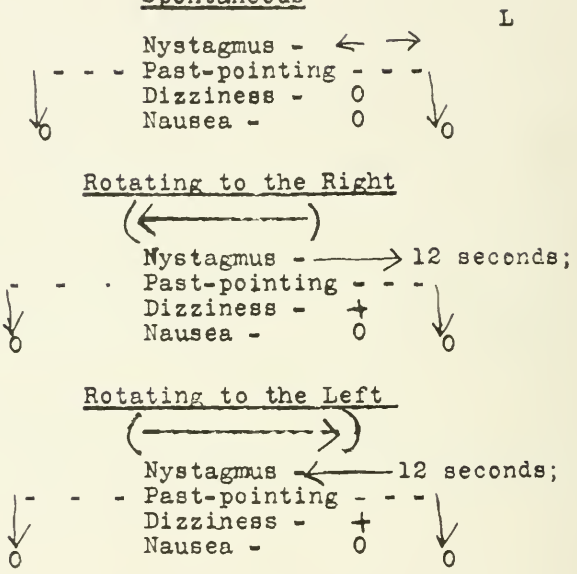
Remained in hospital from November 24 to December 3, when he was discharged.

After leaving hospital he complained of shooting pains from the top of his head to his neck, the pains lasting but a few minutes. Examination revealed dilated veins of the conjunctiva of the right eye, which were not noticed before. O. E. was negative.

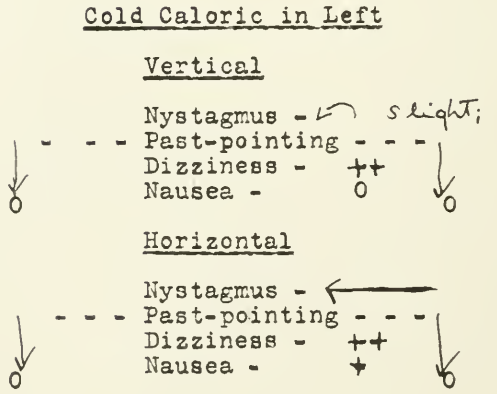
Vestibular Reactions.

Chart 1. J.M.

December 31:



January 27:



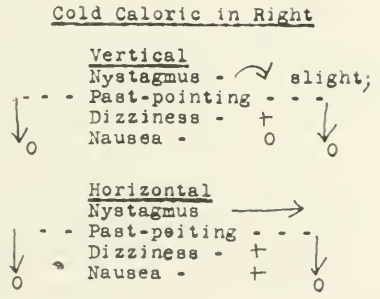
Note on Vestibular Reactions: Cold caloric in left ear gave a nystagmus but no pointing deviation. It seemed impossible to cause a pointing deviation in either arm. Both of the vestibular reactions were very much reduced.

The fact that the reduction on turning is of central, not peripheral, origin, is shown by the observation of Young,* who found that in cases of hereditary deaf-mutism the duration of the nystagmus on turning was not reduced, while in cases of deaf-mutism from a central disease, such as follows cerebro-spinal meningitis, it was not possible to cause an induced nystagmus on turning. Syphilitic deaf mutes may have a normal duration of the nystagmus, or no nystagmus at all, depending apparently upon the site of the lesion, whether peripheral or central.

That the reduction of the duration of the obtainable nystagmus

Chart II. J.M.

February 10:



may be due to intracranial involvement as well as readjusted compensation, and that it may occur immediately upon invasion of the meshes of the subarachnoid spaces by exudate or inflammation are shown by a case of protective or tubercular meningitis, referred to previously, treated by subarachnoid irrigation, rotated on the fifth day after the cerebro-occipito-allantoid irrigation and within a week after the appearance of the meningeal symptoms. In this case, with normal hearing the induced nystagmus obtainable was reduced to 8 and 13 seconds.

*Young, Calvin: Vestibular Reaction in Deaf-mutism. Journal Laryngology and Otology, November, 1921, Vol. 36, No. 11, p. 524.

Griffith (Laryngoscope, January, 1920, p. 22, and March, 1920, p. 129), demonstrated experimentally what had been clinically proved by Dunlap (Jour. A. M. A., Vol. 73, No. 1, p. 54), that repeated turning reduced and finally abolished the induced nystagmus as well as the vertigo and past-pointing.

Robertson, C. M. (Laryngoscope 32-105, Feb. 22; reviewed in Int. M. Educ. & S. Jour., April, 1922, 85-157) also found reduction of nystagmus on repeated flights and turnings.

It is possible that the reduction occurs only when the virulence of the infection is confined chiefly to the meshes of the subarachnoid spaces of the posterior fossa as in the cases herein reported; while in the case of P (Trans. Amer. Otological Society, 1921) the inflammation following the line of fracture was confined mainly to the subarachnoid spaces of the cerebrum. In this instance, the pus and bacteria found in the lumbar puncture may not have been uniformly diffused over the cerebellar cortex to the extent and degree as when associated with protective and hemorrhagic lesions of the posterior fossa itself.

Conclusions and Deductions:

(1) In cases of increased intracranial pressure there is a reduction of the reactibility of the vertical canals of the side opposite the lesion, as evinced by the cold caloric when applied in the upright position with the head tilted forward 30°;

(2) When the lesion is situated above the tentorium, however, the reactibility, although reduced or delayed, is generally attainable. In increased intracranial pressure of the *posterior fossa* the reduction amounts to a complete absence of reactibility of the vertical canal of the unaffected side;

(3) This vestibular manifestation of increased intracranial pressure—namely, reduction of and in the posterior fossa, absence of reactibility of the vertical canals of both ears to the cold caloric—is a general diagnostic symptom of importance but is of no localizing value. Its presence on the other hand, largely prevents further localization by the vestibular tests alone.

(4) A protective collection of abnormal cerebrospinal fluid with partial obliteration of the cerebrospinal fluid circulatory system pathways from labyrinthine involvement, either suppurative or traumatic, situated over that portion of the anterior surface of the cerebellum behind the posterior surface of the petrous pyramid, and manifested by a protective meningitis—shown by a high cell count but without microorganisms in the lumbar puncture—has a definite vestibular symptom complex.

(5) This vestibular symptom complex is:

(a) Loss of reactibility of the vertical canal of the opposite side, as well as of the affected side, from increased intracranial pressure;

(b) Early reduction of the maximum duration of the induced nystagmus, from stimulation of the opposite side from turning;

(c) The duration of the nystagmus from turning being only about 50% of that obtainable after destruction of one labyrinth, the reduction, therefore, is of intracranial and not labyrinthine origin;

(d) Both vestibular manifestations appear sufficiently early to be of diagnostic value and continue for a considerable period, and in certain cases are permanent;

(e) The accompanying spontaneous past-pointing deviation, from the intracranial involvement, rapidly disappears, probably by the establishment of compensation;

(f) Clinical evidence of the earlier presence of a spontaneous past-pointing deviation can be elicited by turning, as after a considerable period of time—after several years in one of my cases—it is impossible to induce a deviation in the opposite direction;

(g) The permanence of the latter vestibular manifestation —permanent inability to induce past-pointing of an extremity in the opposite direction to a previous spontaneous past-pointing deviation—may account for much of the confusion in regard to pointing deviations in all intracranial lesions, both gross and microscopic; if further observation confirms the suggestion here offered that inflammatory and infiltrating lesions of central portions of the vestibular apparatus sufficient to be accompanied by spontaneous past-pointing deviations possibly are never wholly recovered from—as shown by the absence of induced past-pointing in the opposite direction—while those from pressure such as that caused by tumors, endothelioma, cysts, and extradural hemorrhages, may entirely disappear;

(h) In the presence of a suppurative or traumatic lesion of one labyrinth, absence of reactibility of the opposite vertical canal to cold caloric, as well as of the affected side (associated with a 50% reduction of the maximum attainable duration of the inducible nystagmus from stimulation of the unaffected horizontal canal by turning—with or without an associated spontaneous pointing deviation), is a symptom complex of intracranial involvement of diagnostic value and suggests a protective meningitis with localized serous effusion—situated on the anterior surface of the cerebellum behind the posterior surface of the petrous pyramid near the internal auditory meatus;

(6) As intrapiarachnoid abscess from labyrinthine suppuration frequently is located in this situation, it is suggested that possibly the same vestibular symptom complex may be present here also; in which case—although this suggestion is purely from analogy and as yet has no clinical evidence to support it—this vestibular symptom complex would be of localizing value in distinguishing intrapiarachnoid abscess from intracerebellar abscess.

(7) The induced nystagmus from turning and caloric are fundamentally different because of the difference in the nature of the stimulation;

(8) Consequently, in diseases of the nervous system they should be regarded independently of each other, as they are capable of furnishing separate localizing diagnostic information.

(9) In the cerebellar cortex, or in that portion of it which is covered or reached by the meshes of the piarachnoid, is located a part of the mechanism which has to do with the *duration* of the induced nystagmus by turning. Choking of these meshes by inflammatory exudate (as in protective meningitis from labyrinthitis or from hemorrhage complicating linear fracture of the skull) reduces the duration of the inducible nystagmus immediately by about 50% of the normally obtainable nystagmus from turning. This sign is of great general diagnostic and medico- legal value.

(10) This reduction of the duration of the nystagmus from turning may be a fatigue (non-sustained), a muscular phenomenon, which is now recognised to be a part of the cerebellar functions of asthenia and astasia.

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EPIDEMIC ENCEPHALITIS AUDITORY AND VESTIBULAR LESIONS.

By ISIDORE FRIESNER, M. D., New York City.

The following clinical studies are based upon six cases of epidemic encephalitis in which the manifestations were wholly, or in a large part, due to disturbances of the auditory or vestibular apparatus. Four of these cases were observed by me in the past two and a half years. For the histories of the two remaining cases I am indebted to my colleagues. It must be apparent, at a glance, general conclusions regarding this phase of the disease cannot be well drawn from so meager clinical material, and yet the almost total absence of similar studies in the literature is, in itself, sufficient justification for this survey.

After a fairly thorough search of the literature regarding this disease I have been impressed with the comparative rarity of involvement of the eighth nerve. I have been able to examine the records of 145 cases of epidemic encephalitis at Mt. Sinai Hospital during the last two years. Among these there was one instance of bilateral total deafness. In many the stupor and the consequent lack of co-operation made complete functional ear tests impossible. However, with the exception cited above, there was no other evidence of eighth nerve involvement.

Barker in his report of eight cases, mentioned tinnitus as a symptom but once. Here the ears and presumably both the auditory and vestibular apparatus were normal. Eagleton reports a similar case of tinnitus and normal hearing. In Tilney's twenty cases there is no mention of auditory or vestibular manifestations. In Skwersky's ten cases the ears were normal. In Hall's analysis of sixteen cases there is one with vertigo and tinnitus. Functional tests are not recorded, but the general manifestations of the disease were far more severe than those of any of the cases observed by me. Occasionally one meets with recorded evidences of a disturbance in the vestibular apparatus, as in Alexander's Case II, where a vertical nystagmus upwards was present. Case IV of the series here reported showed a similar phenomenon due undoubtedly to a lesion in the pons. Mills and Wilson also report a case with vertical nystagmus upwards. Alexander and Allen reviewed 182 cases in the

literature, and selected 100 from that number as being typical. In their analysis of the symptomatology of these cases there is no mention made of eighth nerve involvement.

Most of the cases in this series lacked some of the cardinal symptoms of epidemic encephalitis. Thus, only in one instance was there either lethargy or asthenia. Cases of the type reported here, several of them with exceedingly circumscribed lesions, probably represent a very mild, or even an abortive form of the disease. Yet it has seemed that in all the cases the diagnosis was well established. An explanation of some of the cases on a basis of toxicity has been offered. One case was originally reported as an instance of botulism. Hall, however, has pointed out that the Cinderella foot of botulism will not fit the glass slipper of this syndrome, and in the light of our later knowledge this case must be considered an instance of epidemic encephalitis with typical multiple lesions. Toxic agents may cause an endemic, rarely an epidemic; never a pandemic.

CASE REPORTS.

Case I. School-boy, aged nine. April 30, 1920 he complained of tinnitus in the left ear, followed by vertigo, vomiting and difficulty in walking. His temperature was never over 100°. On May 3rd, examination showed no tympanic disease on either side. Normal hearing right; total deafness left. There was slight spontaneous nystagmus to the right and a positive Romberg. The direction of the falling was toward the left, but could be influenced by changing the position of the head. In the interval between May 3rd and May 10th, the date of the second examination, a lumbar puncture was done. The fluid was clear containing no cells, and no excess globulin. On May 10th, falling was no longer regularly toward the left and could not be influenced by changing the position of the head. The total deafness persisted. The left static labyrinth, however, was normally irritable calorically, and there was normal vertigo and normal past pointing following stimulation. On May 19th, with a noise apparatus in the right ear, patient heard 26 double vibrations but not 2048 (C^{d}). On June 5th, with a noise apparatus, he heard a forced whisper: heard 26, and June 26th he heard ordinary conversation. The upper and lower tone limits are normal. The Weber is referred to the head. At this time he showed a peculiarity which has persisted to date, i. e., with a noise apparatus in his right ear, when one whistles with the lips, he interprets the sound as a grunt. Asked to imitate the sound, he reproduces the pitch of the tone accurately, but has no idea of its quality.

Remarks. This is an instance of total loss of function of the auditory branch of the eighth nerve with what was perhaps, in the beginning, a collateral edema of the vestibular branch. The slight, if any, loss of function in the vestibular branch disappeared rapidly. Some hearing returned in about three weeks from the onset of the disturbance. It is now, however, about seven months since the beginning of the attack, and it appears that we are justified in concluding that a total restitution of hearing in the left ear will not occur.

Case II. A jeweler, aged forty-two, never had any previous ear trouble. Was seized suddenly four days ago with tinnitus and deafness in the right ear. Slight pain, no vertigo. There was no tympanic disease. Weber was referred to the left. Rinne, left side plus, right side minus ad infinitum. The hearing in the left ear for voice, whisper, watch, etc., was normal. The tone limits were normal. The right ear was totally deaf. There was no spontaneous nystagmus, no falling, no past pointing. Rotation to the right and to the left was followed by a nystagmus of good amplitude lasting twenty-five seconds. Vertigo and past pointing were normal. Caloric of both right and left labyrinth positive in forty-five seconds. Both the vertical and horizontal canals were active on both sides. Blood and spinal fluid Wassermann negative. Spinal fluid was under moderate pressure, clear, no cells, plus 1 globulin. After the lumbar puncture there appeared a weakness of the right facial of the central type, and right Babinski and Oppenheim. All these symptoms disappeared rapidly, and in July it was reported that the man had normal hearing.

Case III. That there are degrees of disturbances without total loss of function is illustrated by Case III. S. G., married; family and personal history have no bearing on present illness. Patient was in good health up to four days ago. At that time he had an attack of diplopia. A few days later there was some paresis of the facial muscles on the left side. He was tired and drowsy. Spinal fluid was obtained under normal pressure. Cell count was normal, and no excess of globulin was detected. Hearing on the right side was diminished to about one-half of what was present on the left side. The Rinne was positive, and he localized the Weber to the left ear.

Remarks. Taylor also reports a case with a bilateral disturbance of hearing of the nerve type.

Tinnitus is a symptom occasionally met with in lethargis encephalitis. Eagleton, however, has shown that tinnitus may occur in such a case with no demonstrable disturbance in hearing. Grahe, in his studies of the internal ear in lethargic encephalitis, states that in six of the thirteen cases examined, the bone conduction was shortened. It is especially noteworthy that a case with shortening of bone conduction, but otherwise with normal findings, presented a perfectly normal duration of hearing by air conduction. Grahe is under the impression that there is no relationship between this condition and meningeal changes, since shortened bone conduction occurred both in cases with normal cerebrospinal fluid, and in others with pathological changes in the spinal fluid.

Case IV. A dentist, aged thirty-six, was seized with vertigo, nausea, vomiting, deafness in left ear, diplopia and ataxia which confined him to his bed for one week. He had no fever. He was unable to leave his home or to attend to his professional duties for several weeks, so that by the time he was seen during the fourth week of his illness, many of his earlier symptoms had disappeared. The history, in addition to the above was that his blood Wassermann was negative. His cerebrospinal fluid showed no cells, globulin content normal, Wassermann negative. The otological tests were as follows. Weber is referred to the head. Rinne, both sides positive. Schwabach, slightly shortened both sides. The upper and lower tone limits are normal for both ears. Tested with a noise apparatus, both ears hear low conversation readily. With the exception of the slightly shortened Schwabach, the hearing of both ears is normal.

Static Labyrinth. Station is good, very little swaying. There is no spontaneous past pointing in the right arm; the left arm past points two inches to the left. This persists on repeated tests. There is a spontaneous nystagmus of the rotary type directed to both sides, slightly more marked to the right. There is also a vertical nystagmus upward. Rotation to the right is followed by a horizontal nystagmus of fair amplitude, lasting twelve seconds. Rotation to the left is followed by a nystagmus of very good amplitude, lasting twenty-five seconds.

Tests for Vertigo. Rotation to the right; the right arm touches, the left arm past points one inch to the left (spontaneous past pointing). Rotation to the left; both arms past point two inches to the left. Caloric reaction of the right vertical canals positive but faint, one minute and fifteen seconds. No past pointing in either arm. Stimulation causes diplopia. Caloric reaction of the right external (horizontal) slightly more marked than that of the vertical canals. No past pointing in either arm. Caloric stimulation of the right external canal arouses an oblique nystagmus upwards. Caloric reaction of the left vertical canals positive faint, fifty-seven seconds, reaction of external canal more marked. No past pointing in either arm.

Remarks. The hearing is normal. There is a spontaneous nystagmus to both sides and also upward. Functional tests show an interference with all reactions following stimulation of the left labyrinth. Caloric stimulation of the right vertical canals causes diplopia, of the right external canal an oblique nystagmus upwards. The nystagmus following stimulation of the right labyrinth is nearly normal while the vertigo is markedly suppressed. These facts indicate that the lesions are not peripheral, but are situated in the brain stem and probably in the cerebellum as well.

Case V. F. W., physician. Was seized about March 4, 1920 with an attack of vertigo, nausea and vomiting. On March 5th and 6th there was diplopia. The vertigo continued for three days and gradually subsided within a week. Never any fever or headache. At the end of the week he was examined. There was no tympanic disease. The hearing of both ears was normal. There was a coarse rotary nystagmus to the left. In walking he swayed to the right, and supported himself in his right side. Unfortunately no other stimulation was applied to the static labyrinth, except rotation. This was repeated several times, and showed definitely a total loss of reaction, i. e., of both nystagmus and vertigo on rotating to the left, whereas on rotating to the right there was a normal nystagmus lasting twenty-two seconds, with normal vertigo.

Remarks. There may be some question as to the validity of the conclusion that we are dealing here, so far as the ears are concernd, with an isolated lesion of one vestibular nerve. It may be remembered, however, that this patient fell to the right, that he supported himself on his right side, that he had a typical rotary vertigo, with objects moving about to his left and that he had a spontaneous rotatory nystagmus to the left. These facts, with the results of the rotation tests seem sufficient to warrant our conclusion.

Since the above was written, I have had confirmation of the fact that an isolated vestibular lesion can occur in lethargic encephalitis. The following case was observed by my Associate, Dr. Maybaum, in the wards of Mt. Sinai Hospital. The patient was a man who showed a horizontal nystagmus to the right without ataxia or spontaneous past pointing. He had normal middle

ears and normal hearing. Caloric stimulation of left vertical and horizontal canals gave normal responses, but, neither the right vertical nor horizontal canals reacted to caloric stimulation.

In conclusion, it may be stated that lesions of the eighth nerve in epidemic encephalitis are rare. This rarity may be more apparent than real, for Eagleton, in his examination of four cases, states that he was able to demonstrate disturbances in the vestibular apparatus in every one of the four.

The involvement may result in a diminution of, or a total ablation of function. I have had no personal experience with lesions affecting both branches of the eighth nerve followed by total ablation of function, but Mills and Wilson report two such cases.

Either the auditory or vestibular branch may be involved. Judging from the number of cases examined and reported, there is an unusual proportion of vestibular lesions. The virus appears to exercise an exceedingly fine selective action.

Restitutio ad integrum occurs perhaps more frequently in the vestibularis than it does in the cochlearis.

The polioencephalitic cases, with exceedingly limited lesions, undoubtedly represent an abortive type of the disease in which many of the cardinal symptoms of epidemic encephalitis are lacking.

The diagnosis in the absence of other confirmatory findings is based upon the exclusion of middle ear disease, of tumor, the absence of the usual serological findings, and other evidences of syphilis, and finally upon the occurrence of these cases during an epidemic of encephalitis followed by fairly rapid recovery.

DISCUSSION.

Dr. S. D. Ingham, Los Angeles, Cal., said there were many interesting points of neurological significance brought up in this paper. Pathology of encephalitis is to a large extent transient. The sequelae are located in the cerebrum rather than in the brain stem. The vestibular and auditory lesions are transient in character, and are referred to the central nervous system. In one case recalled, of organic lesion of the brain stem with disturbance of one ear, there was diplococcus. In another was a temporal lobe lesion and in one ear the patient was not able to judge the quality of the sounds. The cerebrum interprets the higher reflexes and movements and the cerebellum coordinates these movements. The higher responses can be traced to the nuclear mechanism of the brain stem, which may be modified by pressure or local destruction. To correctly interpret the various phenomena is a very complicated problem.

Dr. Louis Guggenheim, St. Louis, Mo., asked if the boy was able to see the source of the sound. The disturbance was probably due to a Wernicke lesion following encephalitis.

Dr. Friesner (closing) said that the boy was blindfolded. He had no idea of the instrument, but he could interpret quality with the normal ear correctly. One knows nothing of the deafness that occurs with central lesions. The auditory fibers, after leaving the restiform body, pass through the trapezoid body. This region can be affected by patches in multiple scleroses. The transient deafness corresponds to temporary amaurosis. In a few cases there has been tumor reported in one temporal lobe and abscess in the other. That is the only evidence we have of disturbance of hearing secondary to a central lesion. The distribution of the 8th nerve in the medulla is so extensive, that if it were hit hard enough to cause total deafness, death would ensue. It would be of great value if in lesions of the brain stem, accurate tests of hearing could be made. The cases should be studied both by neurologists and otologists.

GENERAL CONSIDERATIONS IN INFLAMMATORY DISEASES OF THE NASAL ACCESSORY SINUSES.

By DUNCAN MacPHERSON, M. D., New York, N. Y.

With the utmost conciseness it is impossible that the topic assigned to me can be covered in twenty-five minutes. I shall therefore confine my remarks more especially to inflammatory diseases of the accessory sinuses, and even then beg your indulgence if brevity is secured at too great a sacrifice of detail. Furthermore, diseases of the accessory sinuses in children have been so comprehensively treated by our chairman, Dr. Dean, and others, that little of value can be added.

From the standpoint of inflammatory diseases, all of the accessory sinuses have features in common, and the same etiological factors frequently give rise to similar diseases in any one or all of the sinuses. The necessity of specializing as against generalizing, becomes marked when we enter the field of diagnosis and treatment. It is probably true that autopsy on adults will always show some form of inflammatory sinus disease; and it would seem that many of the nasal lesions resisting treatment are due to accessory sinus disease.

All sinuses are subject to the following inflammations: (1) Acute catarrhal; (2) chronic catarrhal (hyperplastic); (3) acute suppurative; (4) chronic suppurative; (5) confined suppuration.

Acute catarrhal inflammations of any one or of all the sinuses, when due to a local infection and accompanied by coryza or acute rhinitis, frequently demand similar treatment,—that is to say, elimination through the intestinal tract, the kidneys, and the skin; equable temperature,—best obtained in bed; and inhalations. Where there is interference with the exudate from the sinuses, cocaine applied by the surgeon to the anterior ostia, or adrenalin (where there is no idiosyncrasy) may be used on pledgets of cotton, as an ointment or as a dilute spray.

Cold applications I do not use, though they are recommended by some. Owing to occlusion of the ostia, irrigation of the antra, of the frontal, or even of the sphenoid may be necessary. Where obstructions are present in the nose, contributing to recurrences, they should be removed after the attack has subsided. Diaphoresis by means of a Turkish bath, Dovers' powder,

morphine, etc. is advisable. In recurring attacks, especially, a careful search should be made for dyscrasia of the system of the leland or of both. Too much emphasis cannot be laid on this point, as cutting, burning, spraying, and astringents, will, if used alone, prove unsatisfactory. In obscure cases help may be sought from the internist without in any way lowering the dignity of the rhinologist. Hyperemia by aiding the local circulation is of value. Clothing, climate, and occupation must be taken into account.

Where both sides of the nose are obstructed, irrigations are useful. These may be administered by the gravity method or by means of the Holmes post-nasal irrigator. By clearly and carefully demonstrating to patients the method of using these appliances, the danger of ear complications is not great.

Pain may be relieved by heat or cold,—heat being applied by hot water bags, electric pad, electric light bulb, or cold cautery. Internally coal tar products, benzyllbenzoate, the salicylates, or a capsule containing belladonna, camphor, and quinine hydrobromate,—as advised by Dr. Kyle,—are valuable at times.

Where one side only is involved, irrigation for the relief of pain is contra-indicated, the solution carrying the infection into the opposite nostril. If irrigated through the unaffected nostril, the pain is not relieved.

Acute Suppurations of the Maxillary Sinus.

These are usually preceded by nasal infections or alveolar disease. Treatment should be directed toward prompt cleansing of the antrum. After a trial of the more recently advertised silver salts, silver nitrate, zinc salts, saline, alkaline, carbolic acid, lysol, permanganate of potash, peroxide of hydrogen, argyrol, iodine mercurochrome, etc., I am unable to advocate any as superior to another, but simple saline has given me as satisfactory results as any single remedy.

A couple of years ago a series of cases seemed to clear more promptly by frequent changing of the medicament. My impression then was that the bacteria quickly became immune after acute catarrhal inflammations to any bactericide; but on further observation with another series of cases, I was disillusioned by finding my cases clearing as quickly by making few changes. My general routine treatment consists of prompt saline irrigations and autogenous vaccines. If after four treatments, given on consecutive days, no improve-

ment is apparent, I ventilate freely under the inferior turbinate, punching out a piece of the naso-antral wall, and continue the irrigations. In cases where the general nasal mucosa indicates defective fluid-carrying capacity, a mild astringent,—commonly zinc chloride or zinc sulphate,—seems to be of service. Silver nitrate I find difficulty in using without producing its disagreeable oxidizing effect, even when vaseline, saline, etc., are used as preventives. One case of streptococcus hemolyticus infection improved while using sodium salicylate, administered for an intercurrent attack of tonsilltis. The salicylate was continued, and the case cleared up in six weeks. A second case of streptococcus hemolyticus cleared up with the same treatment where no acute tonsillitis was present; but a third, while improved, is still under treatment at the end of ten weeks, receiving two to four antral washings weekly, and I believe will recover without more radical operation than free ventilation. All of these cases received autogenous vaccines.

Out of seventeen cases at present under treatment in my office, fourteen cultured staphylococcus aureus; one, streptococcus hemolyticus; one, Friedlander; and one, bacillus coli communis. I am unable to decide whether the vaccines are of any value. Irrigation, followed by suction, applied by means of a canula inserted through the inferior meatal opening, supplemented by a powder of stearate of zinc or bismuth, helps at times to dry up the discharge.

Chronic Catarrhal Inflammation.

Chronic catarrhal inflammation is rare, the acute form being more likely to be productive of an acute or chronic suppurative type, but it does occur and may demand operation. The diagnosis is obscured by the pathological involvement of the nasal mucosa. Nasal discharge,—watery or mucoserous,—is present and the antrum may require irrigation or even curetment.

Chronic Suppurative Maxillary Sinusitis.

Under treatment these cases sometimes cease to discharge pus quite as promptly as the acute types. This, however, is exceptional. Transillumination and the radiograph showing undoubted thickening,—where no post-operative ostitis or periostitis accounts for it,—I irrigate four or five times, and if the discharge is not distinctly less after the fifth as compared with the fourth treatment, I ventilate widely under the inferior turbinate, as advised some years ago by,—I think,—Dr. Yankauer, and

continue the irrigation for a variable period. Should this be unsuccessful, one of the several forms of radical operation is necessary.

Emphasis is placed on the teeth as a possible etiological factor. Radiographs should be made in all sinus cases, even where pus has been clinically demonstrated. They serve not only for diagnosis but as an aid to treatment, revealing bony septa in the sphenoid, frontal and antra, and may demonstrate necrosis, tumors, and other complications. The relative skill of the surgeon is not sufficiently considered when advocating certain surgical operations, for one man may get brilliant results where another would fail.

When frontal sinus pain is accompanied by pus in the middle meatus, exploratory irrigation of the antrum is advisable, since it frequently not only gives relief from the pain but accelerates healing of any suppurative frontal involvement that may be present. Here, too, a radiograph may be helpful in revealing a frontal bone without a sinus.

Ethmoiditis.

The classification of ethmoiditis is confusingly elaborate, whether approached from a pathological hypothesis, clinical history, or both. Ethmoiditis may be latent; it may be manifested by obscure objective symptoms; or it may be attended by free pus discharge; it may be obscured by polypi in the nasal fossae, or complicated by orbital abscess, meningitis, or local caries. The diagnosis must often be reached by exclusion. For clinical purposes, ethmoiditis may be classified as acute non-suppurative, and acute suppurative; chronic non-suppurative, and chronic suppurative.

Formerly it was believed that an acute infected ethmoiditis, unless going on to suppuration, subsided with the general acute rhinitis of which it was a part. Now, it is a well established belief that a residual ethmoiditis may persist and continue to advance long after the turbinal mucosa and the mucosa of the meatuses are graded as macroscopically normal, and the light of the normal microscopic findings of tissue removed from the ethmoid in cases accompanied by intercurrent optic nerve involvement, it would seem that the nasal mucosa itself, if submitted to microscopical section, would measure up to the accepted standard of normality, differing not at all from sections taken from ethmoids that are accompanied by complicating optic neuritis,

for instance. (The term, optic nerve involvement, is used here owing to the clinical difficulty of differentiating between papillitis, papilloedema, choked disc, and optic neuritis.)

The treatment of acute catarrhal ethmoiditis is similar to that of general acute catarrhal sinusitis.

Chronic Non-suppurative Sinusitis.

This class of cases presents special symptomatic diagnostic difficulties, and by some observers is, unfortunately, placed for purposes of treatment, in the same category as the suppurative type, in which latter the optic nerve changes are more rapidly destructive, requiring more active radical treatment. Widely different methods are practiced in the treatment of these hyperplastic cases where optic nerve involvement is present. A greater divergence of opinion exists relative to the proper handling of this subdivision than in any other. In the hands of some rhinologists, the treatment consists of immediate and complete exenteration of the ethmoid labyrinth; by others, vacuum treatment is first employed in an attempt to save the ethmoid cells; while still others open or aerate the posterior ethmoid cells, and perhaps the sphenoid. In a certain percentage of these cases it is admitted that the optic nerve involvement disappears without any treatment, local or constitutional and it is fair to assume that the total of cures attributed to any form of treatment includes a number of such cases. It is claimed by some that all cases recovering as the result of vacuum treatment would recover by radical operation, that no time is lost, and that danger of complete loss of vision is lessened. I have yet to see a case that I would class as hyperplastic ethmoiditis, suffer from the postponement of radical surgery, for twenty-four to seventy-two hours, while simple vacuum treatment was being carried out under the close observation of a competent ophthalmologist. I have seen the dura exposed, meningitis supervene, and know of one case where the optic nerve was cut when the radical operation was performed. I therefore feel that ethmoidectomy is not to be lighting advised. We all know that the public is not always served by competent operators, and that where equally good results follow simple procedures the public is thereby safe-guarded. I believe therefore that in all cases of hyperplastic ethmoiditis, where aeration or local blood-letting only is the object of the operator, the patient will not only not suffer, but that his interests will be better served by first

applying vacuum treatment. In many cases, two or three vacuum treatments serve as a guide to the advisibility of continuing that treatment, the beneficial results being promptly evident in most instances. In one case where decompression had been advised, the ophthalmologist, Dr. Bernard Samuels, reported the patient's vision was limited to the counting of fingers. After one vacuum treatment the vision was increased to 10/200; in one month, it was 20/20. In my experience, recurrences are not frequent; but if there is a recurrence, more radical measures may always be resorted to. Even here, however, I do not believe one should advise complete exenteration before first having recourse to opening the posterior ethmoidal cells, as advocated by Dr. White of Boston, and others. Excellent results follow. To this procedure there is relatively slight objection. In all such cases, as a matter of routine the neurologist and the otologist must be consulted in order that intra-cranial pressure be not overlooked. In hyperplastic cases, however, the ophthalmologist is as yet the chief guide to operative surgical interference,—and by the term surgery I mean conditions remedied by instrumental appliances.

In the type of ethmoids under consideration, radiography is an aid, inasmuch as it confirms the diagnosis of non-suppurative ethmoiditis already made, or it may reveal a few cells so impervious to the ray as to compel further careful examination, or even small areas of suppurative involvement, which have eluded clinical examination. Nasal deformities interfering with drainage, should of course be corrected.

As a rough guide, some ophthalmologists place about 3 diopters as the limit of disc projection produced by ethmoiditis.

Acute Suppurative Ethmoditis.

This is frequently accompanied by sphenoidal, frontal, and antral involvement, or by any one of them, or the suppuration may be confined to one group of the ethmoidal cells. If untreated, it tends to chronicity,—a sequel that treatment does not always prevent,—although cases may spontaneously recover. Nasal abnormalities causing obstruction should be corrected, bearing in mind in the event of septal resection the danger of infection and abscess between the flaps and the advisibility of postponing the resection until a later date if possible. Nasal irrigation directed from the opposite side and postnasal irrigation by means of the Holmes post-nasal irrigator or spray are

helpful, the patient always being directed to clear the nose of the excess of fluid without closing either side. Suction treatment, as advised several years ago by Dr. Haskin, and vacuum treatment,—the latter used in my hands without alternating pressure and medication,—has served me well. Breathing medicated vapor is also a valuable routine treatment; it frequently allays pain, and reversing the cone so as to steam the parts externally is helpful.

Internally, coal tar, salicylates, opiates, benzylbenzoate, etc. are recommended.

If despite these measures suppuration continues, the involved cells should be exenterated, and in doing so too much emphasis cannot be placed on the care necessary to avoid the opening and infecting of a healthy sphenoidal sinus.

Chronic Suppurative Ethmoiditis.

In this disease where polypi are not present, vacuum and suction treatment afford much relief, and in a proportion of cases the suppuration ceases. Relief is obtained after the first or second treatment, while the cure may be postponed for months. Vacuum treatment is peculiarly applicable to cases in which cutting is contra-indicated, such as hemophilia, in the aged, etc. In the case of one hemophilia under my care, in which the ethmoid, sphenoid, frontal and antra were diseased, suppuration disappeared at the end of eighteen months. In this case, the antra were irrigated in addition. Another bleeder, suffering from pan-sinusitis, I have now had under treatment for more than a year; both antra were involved, and bleeding was so profuse after trocar and canular washing that I feared the results of ethmoidectomy. At present one antrum has remained clear for several months, while the other is being washed by the patient, and shows a cast about the size of an apple seed every third or fourth day. A small crust appears on the opposite side in the region of the anterior ostia which leads me to believe that the process is slumbering in the anterior ethmoid cells. Generally speaking, however, I believe the better treatment consists in the removal of the infected cells. It should be borne in mind that residual inflammation follows both kinds of treatment, and is, therefore, not a just criticism of vacuum treatment alone. It is also stated that atrophy of the optic nerve follows vacuum treatment. My experience is that where the nerve has been involved for any considerable time, atrophy follows any

form of treatment. In ethmoiditis co-existent with atrophic rhinitis the field is usually so bloodless under local anesthesia that one can follow the disease from cell to cell, rendering a complete ethmoidectomy unnecessary. It should be borne in mind that where the antrum is acting as a reservoir only it should not be radically operated upon. Sometimes one sees operators who probe the frontal sinus as a matter of routine procedure after ethmoidectomy. Unless the frontal sinus has been shown to be infected the danger of this practice is apparent. Where polypi are present, either in the nasal fossae or in the ethmoidal labyrinth, they should of course be removed. Cutting operations of the ethmoidal sinuses on adults are more easily done under local anesthesia. Orbital abscesses should be treated as an abscess in any other part of the body; that is to say, evacuated, but with the minimum of trauma to the cells compatible with good drainage. Several years ago our Chairman called attention to the relationship existing between the odor of decomposition and scabbing in cases where the antra and sphenoidal cells were suppurating, and stated that a diagnosis of ozena was frequently made when such conditions were present. In such cases the result of treatment directed to these sinuses is frequently surprising and gratifying.

Frontal Sinuses.

Acute Catarrhal Type.

The general treatment of this disease does not differ from that recommended for similar inflammation of the other sinuses. General treatment takes precedent over local. In cases where pain is due to negative pressure relief is sometimes obtained by treating the mucosa with cocaine or adrenalin; by breaking and bending the middle turbinate toward the septum if there is sufficient room. Resection of the anterior end of the middle turbinate and breaking down the ethmoidal bullae are also practiced. Hot irrigation and external heat or cold are helpful. Avoidance of surface chilling is more important in cases of acute sinusitis than in any other disease. Washing out the sinus is indicated if symptoms do not subside. This may be done by resecting the anterior end of the middle turbinate, or by opening the naso-frontal canal in the region of the ager nasi cell as pointed out by (I believe) Dr. Mosher some years ago. Obtaining effective drainage should not be too long delayed, as chronic thickening will predispose to recurring attacks which

sooner or later will become purulent. Coal tar products, the salicylates in some form, opiates, benzylbenvoate, chloral hydrates etc., are used to allay pain. The tendency of this disease is to spontaneously recover. Where this does not occur the acute purulent is the most likely type to supervene.

Chronic Catarrhal Frontal Sinusitis.

Here drainage is usually the prime factor of treatment. Polypi and hypertrophus of the middle turbinate should be removed and deviation of the septum corrected.

Acute Suppurative Frontal Sinusitis.

Here again drainage is imperative, and is attained by vacuum treatment, anterior middle turbinotomy, breaking and forcing the middle turbinate toward the middle line, removal of obstructions and opening in the region of the ager nasi or bullae. Hot irrigations for the relief of pain in unilateral cases, as pointed out by Skillern is to be deprecated, the disease being carried to the opposite side. Hot irrigations applied to the unaffected side are not serviceable for the relief of pain. All patients suffering from this disease should be carefully safeguarded against exposure and for this reason should be treated in an institution, or when preferable in the patient's home. Riding back and forth daily to and from the doctor's office increases the danger and renders the disease clearly more intractable. External and internal treatment recommended for general sinusitis is applicable here. I recall but three cases of primary attacks in which under this treatment an external operation was necessary. Septal resections during the acute suppurative stage should be avoided if possible, owing to the danger of an infection. Two cases of my own operated upon during the past year became infected from this source. Nevertheless at times this risk should be assumed by the surgeon.

Chronic Suppurative Frontal Sinusitis.

In cases not previously operated upon a radiograph is of more value than where post-operative changes fog the picture. But even here a picture may reveal bony septa, or even absence of one or both sinuses in addition to confirming the clinical findings. On two occasions, however, once about ten years ago and again about three years ago, persistent pain, and a small amount of mucopus, but chiefly radiographic findings, led me to make an

external exploratory opening. In both cases I found normal mucous membrane lining the sinuses. If on examination we find obstructions to the outlet due to septal deformities, turbinal hypertrophies, polypoid changes, or degenerated anterior ethmoid cells, they should be removed. Where possible lavage of the sinus is to follow the removal of the obstructions, we should enter the sinus with a probe to assure ourselves that no further cutting will be necessary to achieve this object. Irrigation of the sinus carried out immediately after the removal of the obstructions brings pus in contact with a large area of newly lacerated structure, and is not free from danger. One death from meningitis came to my notice some years ago following lavage of a maxillary sinus after instead of before a turbinectomy. Lavage may be carried out every day, or less frequently depending on the amount and character of the discharge and the judgment of the surgeon. The solutions, too, vary as in maxillary sinus disease from salines to antiseptics and astringents. In cases where I have been unable to decide whether there was marked pathological change in the lining of the sinus, I have continued irrigations for months, and have obtained as satisfactory general results as from the radical external operation The Ingalls method I have not used. Enlargement of the frontonasal duct should be resorted to where removal of all obstructions interfering with drainage, and removal of the anterior ethmoid cell followed by lavage, fail to give relief. A radiograph assures greater safety, no matter what operation is to be performed. Thompson's method of using a grooved rasp fitting over a small probe placed posteriorily is less dangerous in unskilled hands than the other intranasal methods, and is the one I prefer, Failing to obtain the necessary results by the intranasal route, the external must be resorted to. The necessary result is said advisedly, because the external radical operation is, in many cases, a failure, and may require to be repeated a second or a third time.

Last week I saw a patient who had been operated upon five times,—twice by myself. This (the fifth) time the wound was left open to granulate from the bottom. Here new bone formation of ivory density completely occluded the nasofrontal duct, crossing from the orbit to the nasal septum.

But with frequent headaches, bony septa in the sinuses preventing drainage, caries, or necrosis of the sinus wall, danger

or presence of intra-cranial complications, and profuse discharge, the case demands an external operation.

Despite some failures, the Killian, or some modification of the Killian, is the method which gives me the best results. In suitable cases the operation may be more cleanly done, and with much less hemorrhage (an important consideration) under local anesthesia. The Lothrop operation has been a distinct aid in two or three of my stubborn cases.

Inflammatory Disease of the Sphenoidal Sinus.

Acute non-suppurative; chronic non-suppurative; acute suppurative; and chronic suppurative.

Acute Non-suppurative Sphenoidal Disease. I have never recognized a case of acute catarrhal sphenoidal disease existing as a distinct entity, but by inference we may assume that it co-exists where the same condition is present in other sinuses. Its treatment follows the same general lines; that is to say, constitutional treatment.

Chronic Non-suppurative Sphenoidal or Hyperplastic Disease. The existence of this disease independent of involvement of the ethmoidal sinuses, is probably rare, at least I have never been able to demonstrate it. The sinus is so inaccessible and the nasal mucosa so obscuring that inspection is difficult in the majority of cases where the necessity is greatest. Despite the number of authentic cases that have been quoted, no case has come under my observation where I could be certain that hyperplastic sphenoiditis alone caused optic nerve disease. This may be due in part at least to the fact that in this class of cases (where pus is not demonstrated anteriorly or posteriorly in the nasal fossae or in the sinus) the treatment given consisted of suction and vacuum, and was therefore applied to the sphenoid as well as the ethmoid.

Acute Suppurative Sphenoiditis. Where the diagnosis is assured and in the absence of urgent complications, lavage and vacuum and suction treatment are indicated, frequently giving satisfactory results, relieving the discharge and the headache. In fact either one alone will occasionally do so, although in what percentage of cases I cannot say, as my records are not compiled and are not sufficient in number to act as a basis for a statistical record. The ethmoidal sinuses, especially the posterior, always need careful examination, even in cases where the sphenoidal ostia are easily seen and accessible. As in frontal sinus disease,

where the radical operation does not always result in cure, so opening the sphenoids in sphenoidal disease does not always result in cure, the opening having a tendency to close with granulations and fibroid change. Hence, in chronic or acute uncomplicated suppurating sphenoidal sinus disease lavage, suction and astringents applied about the ostia should receive a fair trial before the radical operation is done.

Chronic Suppuration of Sphenoidal Sinus. I believe the vast majority of cases will require enlargement of the ostia more or less radically done, and where any opening is needed a large one is to be preferred. Downward and inward are the safer directions to cut; always being careful not to permit the instrument to jump or slip when the spring is opened or closed, and being careful to keep away from the walls of the sinus, especially the superior and external. Post-operative treatment may in certain cases consist of occasional cleaning only; in others, frequent cleansing with the application of astringents to the granulations, preferably silver nitrate, may be required.

As stated when referring to the application of vaccines when applied to treatment of disease of the other sinuses, I believe that if they do no harm and may do good, we are justified in using them.

In rapidly destructive cases occuring either in acute or in certain exacerbations of chronic suppuration, no time should be lost in evacuating the pus; as in ethmoidal cases with optic nerve involvement, the ophthalmologist should be in close attendance.

Bi-lateral Chronic Pan-sinusitis.

My custom here is to operate on one side, wait for the patient to recuperate, and then do the second. The shock from hemorrhage and anesthesia, together with the length of time necessary for the average man to complete both sides, is a greater strain than many patients can survive.

Using a posterior plug, I first open the antrum, then the frontals and ethmoids and sphenoids. It is difficult to become oriented in Jansen's operation, and I have not done it for ten or twelve years, preferring a combination of the Caldwell-Luc and Killian.

Summary.

After all has been recorded in an attempt to outline the general method of treatment grouped in our sub-divisions, it cannot

be gainsaid that many cases respond in an irregular way and will be found to respond to no fixed routine rule. Vaccines seem to help in some cases, and to be useless in others. Why these varying reactions to treatment manifest themselves, I do not know; nor do I know anything that will serve as a guide to treatment better than the judgment and experience of the individual surgeon.

DISCUSSION

Dr. Harris P. Mosher, Boston, said that he was in sympathy with an attempt to classify the anatomy and surgery of the sinuses. The paper was very comprehensive. The speaker said he had never been able to use vacuum treatment successfully. He was glad Dr. McPherson recommended exhaustive therapeutics before resorting to surgery, and that the sinus was not opened unless there was reasonable suspicion it was diseased. He mentioned opening orbital abscess under cocain; that should be done under general anesthesia. In frontal sinus cases the modified Killian gives the best result. The full Killian is only used in large sinuses. The X-ray helps in deciding whether one should do an intranasal or do an external operation. If there is a short anteroposterior diameter, one can't get much success working through the nose. He would end with one caution to the younger men who are apt to be more impetuous. Be sure to respect the virginity of the nasal frontal sinus.

Dr. W. B. Chamberlin, said that the procedures were determined to some extent by the economic status of the patient. In one case, a well to do woman, who said she was willing to spend the time coming to the office, he cleared up the case by conservative measures, which consisted of 27 irrigations of the antrum. She never had return of the trouble. In cases where the patient had to be quickly cured and return to work it was better to relieve the symptoms by operation.

Dr. F. L. White, Boston, said that many cases are relieved by simple measures, such as the argyrol and adrenalin spray, without resorting to exenteration of the cells. Care must be taken not to syringe infected material from one sinus to another. Cases with optic nerve involvement can get considerable improvement of vision from conservative measures, and were able to go to work

Dr. S. H. Hedges, Charlottesville, Va. said that he did not believe cases with optic neuritis should go to work. They were usually very sick patients.

Dr. R. H. Craig, Montreal, Canada, said that he remembered a case of a lady, 35, with blindness associated with pain on the left side of the head. The Wassermann was negative. Opening of the sinus had no effect on the headache and blindness. Her husband had been operated 15 years before and had then a specific history. It was decided to give the patient iodides in spite of the negative Wassermann, and the blindness cleared up very promptly. If after opening and ventilation of the sinuses, blindness persists, iodides may be useful.

Dr. Coffin (Chairman) said that he hoped Dr. Mosher would be convinced when he said that he had better results from vacuum treatment and the patients seemed more satisfied than with operative work. He used all the usual instruments, and forceps, but would part with any of

them sooner than the suction apparatus. He cited two cases of patients, (both artists) who had been very nearly blind, and regained nearly normal vision after suction treatment. One patient, who insisted on an ethmoid operation besides, did not do so well after this as after the vacuum method, her vision dropped down, and she got discouraged and was lost sight of.

Dr. McPherson, (closing) said that he did not use local anesthesia for orbital abscess. One point he would mention was that cleaning of the antrum should be preceded by cleansing of the nose, or one would get an antral infection from manipulation. He certainly agreed with Dr. Mosher that the frontal duct should be respected.

MEETING OF THE EASTERN SECTION, HELD IN BUFFALO, NEW YORK, FEBRUARY 11, 1922, UNDER THE CHAIRMANSHIP OF DR. JOHN F. FAIRBAIRN, OF BUFFALO, N. Y.

SOME REMARKS ON THE INDICATION FOR THE SIMPLE MASTOID OPERATION IN THE ADULT.

By JOHN B. RAE, M. D., New York City.

We arrive at a diagnosis of mastoiditis on information which we receive from three sources (1) The General or Constitutional Symptoms. (2) The Information which we receive from the Ear itself. (3) The Laboratory aids.

The temperature in uncomplicated mastoiditis is low and may be normal even in the worst cases. There is general malaise, loss of appetite and coated tongue. Pain may or may not be an evident symptom. When present it is referred to the temporal region, is dull and aching in character, is worse at night and sometimes disturbs or prevents sleep. Pain may also be entirely absent.

On inspection of the canal and drum head, we will find an acute or chronic otitis media or there will be evidence or history of a recent involvement of the middle ear. It is generally conceded that a purulent middle ear always precedes a pus mastoid even although the history may be indefinite and the local appearances little suggestive. The location and size of the perforation in the drum head, should that be present, will be noted and its adequacy or inadequacy for drainage will be considered. An effort should be made to estimate the amount of discharge. This is done by wiping out the canal. The patient is then directed to Valsalva and the amount of pus forced through the opening into the canal should be noted. This procedure may be repeated until the middle ear cavity is emptied and in this way an estimation of the amount of discharge may be arrived at, at one sitting. This estimation of the amount of discharge is important as in the absence of other symptoms this may be the deciding factor in determining operation. Acute suppression of discharge with increase of pain and temperature is also quite significant. Presence or absence of prolapse of the postero-

superior canal wall should be looked for. This is an edema at the junction of canal wall and drum head. The acute fold at the junction is lost and it is impossible to say where the canal wall terminates and drum head begins. It is most likely due to swelling of the periosteum caused by involvement of cells in the neighborhood of the posterior canal wall or of the canal wall itself.

Swelling behind the ear is a late symptom of mastoiditis in the adult. This swelling may be simple edema or subperiosteal abscess. Needless to say in either case this would be an indication for operation in the adult. Swelling behind the ear is not always due to mastoiditis and will call for a differential diagnosis between that due to mastoiditis, to furunculosis and to local cellulitis.

When slight in amount post-auricular edema is detected by standing behind the patient and observing both ears at the same time. Obliteration of the post-auricular fold can be thus observed even though displacement of the auricle is scarcely perceptible otherwise.

Perhaps the most constant symptom of mastoiditis is tenderness on pressure. This is elicited by firm pressure inwards and backwards over various points of the mastoid process. Care must be taken that no movement of the cartilage is made as this will cause great pain if we have a furuncle in the canal. Tenderness on pressure is due to direct compression of the inflamed bone and not to periostitis which is a late manifestation. Tenderness is as a rule an early symptom. The extent of the tenderness is mainly determined by the character of the mastoid. It is elicited over the antrum, over the tip and over the whole surface of the mastoid if this is very cellular and involved. It is a mistake to speak of tenderness over the mastoid emissary vein because we can never be certain of the location of this vessel. Tenderness on pressure may be entirely absent. This may be due to an excessively thick cortex, to a very forward sinus leaving little space for cells or to sclerosis of the entire mastoid process.

The Laboratory aids have reference to the identification of the infecting organism, to the total and differential blood count to the blood culture and to the X-ray findings.

While it may be of some value to know the causative organism, too much stress should not be laid on this. Clinical observation

of the progress of the disease is much more important. If we presume for example that a staphylococcus infection is necessarily a mild one we may adopt an attitude with regard to a particular case that is not at all justified by its clinical course. It is the experience of most of us that any infecting organism may in any individual case give rise to the greatest destruction. It is therefor wise to emphasize the importance of clinical observation while by no means minimizing the value of the laboratory aids.

The blood counts and culture are not of any great value in uncomplicated mastoid cases. It is only when the deeper structures are involved or threatened that they are really called for.

It is equally true that there is usually no need for X-ray examination in the average case of acute mastoiditis. Apart from the information which the picture gives to the observer, the exhibition of the X-ray plate to the patient or his friend may have considerable influence in procuring permission for operation.

In the X-ray examinations there should be the close co-operation between the Otologist and the Radiologist. The latter should receive from the Otologist sufficient information about the patient to give him an intelligent idea of the clinical situation. The picture having been taken, both observers should make a record of their interpretations and these should always be checked up in cases which come to operation. In this way a just estimate of the value of this aid to diagnosis will be reached and both clinician and technican enabled to interpret the pictures to the advantage of the patient. We have not yet arrived at the point at which pictures can always be absolutely interpreted and too often do we find that the report is ambiguous and undecided in those very cases in which we had hoped that the X-ray might prove to be a deciding factor and enable us to set a definite course.

With all or a majority of these many symptoms present the diagnosis of mastoiditis is not difficult nor can the indication for operation be missed when they progress or even continue without increase, over a period of a few days. If asked to state in a word the indication for operation the writer would answer that operation is indicated as soon as we are assured of the presence of pus in the mastoid. He can see no reason for delay once that conclusion has been reached.

It must be remembered that in a number of cases in which we have to deal with a mastoiditis complicating an acute middle ear a distinct improvement occurs in all the symptoms when a myringotomy has been performed and treatment started with the patient at rest in bed. Pain subsides, temperature approaches or reaches normal and the mastoid operation may have to be recommended in spite of this apparent improvement.

We must also be on our guard against the natural tendency of a patient to minimize or hide tenderness on pressure. This applies particularly to observation cases in hospitals who have had the chance to see other patients returned from the operating room and to witness dressings. They are anxious to avoid operation and will sometimes do their utmost to delude themselves and deceive the examiner.

In addition there can be no doubt that in unoperated cases of mastoiditis the patient may rapidly acquire a complete tolerance. It is not an uncommon experience to find a patient who has been attending to his every day affairs over a period of weeks without especially complaining of his mastoid which has been slowly going from bad to worse.

It is in the cases of quiet mastoiditis in which most of the classical symptoms are lacking, that the judgment, firmness and courage of the otologist will be called for. With the temperature normal and the patient free from pain the operative indications may be very limited in number and apparent and urgent only to the otologist and not at all so to the patient and his friends. Needless to say the quiet case is particularly urgent because of the danger of the onset of complications during a period of procrastination which to the unskilled observer seems absolutely justifiable. The X-ray here offers its best field of usefulness and if the findings are positive immediate operation should be urged.

The clinical evidence in these quiet cases has to do with the appearance in the canal, the amount and character of the discharge, the presence of persistent tenderness on pressure or less frequently the persistence of an otherwise unaccountable temperature. It is also quite noteworthy that the tongue often remains coated. With regard to the canal we find marked prolapse of the postero-superior canal wall or more frequently a progressive narrowing of the entire fundus. The visible drum area is quite small and if discharge be present it may show as a small pulsating bead at the inadequate opening in the drum

membrane. Parenthetically it may be stated that these are the cases in which repeated myringotomies are sometimes performed. It is a safe general statement that when one or two myringotomies made under favorable conditions fail to secure adequate drainage and bring about relief that opening of the antrum is a really conservative measure and that this is indicated rather than further myringotomies.

In those cases in which excessive amount of discharge is the characteristic symptom the pus may be thick and creamy and adhering to all the canal walls so that it fills the lumen of the speculum when introduced. When wiped out the canal will fill up again immediately when the patient is directed to Valsalva. We conclude that this amount of pus is not coming from the middle ear alone.

Tenderness on pressure when present does not differ in location or degree from that described a few minutes ago, and depends on the character of the mastoid itself and the extent of involvement.

We must not forget that in the quiet type being described the decision for or against operation may have to be made not in the presence of all these groups of symptoms but in the presence of only one. And also that the general condition and feeling of the patient are such that the gravity of the situation is by no means apparent to the patient himself, to his friends or even to his general medical adviser.

In conclusion then, in the average case in which all the classical symptoms continue or increase for a few days after myringotomy and treatment, we may be reasonably certain of the presence of pus in the mastoid and advise operation.

It is the opinion of the writer that no definite limit in days should be set down as a safe observation period but that each case should be considered on its merits, particularly from the strictly clinical standpoint, and with regard to increasing or decreasing severity of the symptoms and limitation or extension of the pathological process in the bone. The average case allows plenty of time, say up to a week, for observation in the absence of any urgent call, and experience proves that cases so held over do better after operation when nature has had a chance to collect the constitutional and local resources in the fight against the disease.

In the quiet cases an X-ray is immediately called for. Should this show bone destruction or be otherwise positive immediate

operation is indicated. The contrast between the healthy and the diseased side may prove to the patient that his condition is more serious than he had imagined. It is a constant source of astonishment to find the amount of destruction that has taken place in these cases with a minimum of symptoms.

Should the X-ray be indefinite operation should still be urged.

It is often observed in these doubtful cases that the hearing is diminished out of proportion to the apparent lesion and this is of considerable diagnostic value.

It seems to the writer that the correct attitude of the otologist in face of a quiet mastoid should be to ignore the negatives, to dismiss from his mind what is lacking in the symptomatology and to concentrate on what is positive, even should that be but one lone symptom.

The writer has yet to regret opening a single quiet mastoid in which the average observer has made the clinical diagnosis.

TREATMENT OF ACUTE SINUS INFECTIONS.

By LEE M. HURD, M. D., New York City.

There are a small number of cases, a fraction of one per cent. that, because of the severity of the infection which has or will extend beyond the sinus limits, thereby producing grave complications, require prompt and fearless surgical intervention.

There is a fair sized group which become chronic unless properly treated, but the great majority recover without attention or they undergo medical treatment or minor surgical measures.

The cardinal symptoms for which the patient seeks relief are pain, pus, pyrexia and swelling, in the order named. Many times they are treated by home remedies or by general physicians for neuralgia or, if influenza is epidemic, the case with headache and fever is treated for influenza, even if the nose is occluded and discharging pus.

General Considerations..

For Pain. Heat, electric light, diathermy or hot applications, such as poultices, are used. The electric light can be applied in the form of the Killian Box, the therapeutic light or, which is quite as efficient and much more simple, the ordinary drop-light hung over the patients head so that they may get heat and light at will. *Drugs*—Aspirin, the Coal Tar derivatives and Opium derivatives, are prescribed.

For Congestion. Cocain is highly recommended, but really is irritating and extremely dangerous to give the patient as a spray; adrenalin, also highly recommended, but its benefits are counteracted by its reaction; nitrate of silver in small doses of ½ to 1% as a local application, after the first slight irritation, gives more lasting contraction than either cocain or adrenalin; argyrol, of doubtful service, and hot saline douches. Of the four agents, I depend mainly on the saline douche and nitrate of silver.

For Pyrexia and External Swelling. Surgical relief by drainage.

A rough working rule is, that of the three symptoms, pain, fever and external swelling, if you have any two of them, and surely if you have all three, the case is beyond medical measures

and requires prompt surgical intervention. This, like all working rules, has its exceptions. But, if the infected process has extended beyond the sinus limits, usually into or about the orbit, producing cellulitis, abscess, caries, osteomyelitis, ocular symptoms or cavernous sinus symptoms, though these conditions are very rare, they require prompt external operations for the relief of pressure and drainage of the sinus and the infected area.

Treatment Directed to Particular Sinuses.

Acute Ethmoiditis. The medical treatment outlined above under general considerations meets the requirements of acute ethmoiditis and very rarely, indeed, is surgery indicated. Then, usually, because of an extension of the infection through the sinus walls into the orbit or threatening the anterior cranial fossa, which would lead me to do an external operation by the orbital route.

Acute Frontal Sinus Infection.

The usual medical measures to reduce the congestion within the nose, with suction to reduce the amount of pressure within the sinus, will usually suffice. If there is the slightest evidence of swelling of the lids or an elevation of temperature, it becomes necessary to remove the anterior portion of the middle turbinate and break down the anterior ethmoidal cells which form part of the floor of the frontal, to establish drainage. It is possible only in a few cases to catheterize the frontal sinus without this surgical measure. If it does not promptly relieve the temperature and swelling, it becomes necessary to invade the sinus through the eyebrow, but this is extremely infrequent and it is done to relieve such conditions as orbital cellulitis, osteomyelitis and caries.

There is one type of acute frontal sinus which is characterized by a periodic neuralgic pain which comes on at nearly the same time in the morning and as suddenly ceases about the same time in the afternoon, and the patient feels quite well from that time until the next morning. I have no satisfactory explanation for this type; though they suffer the most pain, they are not inclined to be as serious as the more common type.

If the pain is not too severe and does not extend over too many days, I try by the aid of local depletion, suction and diathermic heat externally, to evacuate the secretion which is

of a thick mucoid character, rather than purulent, because as soon as this thick collection is removed from the sinus, the pain disappears. I have demonstrated this in several severe cases by enlargement of the frontal orifice and douching out this mucoid plug and had pain promptly disappear.

The sphenoid sinus, besides the general measures, can usually be catheterized and the contents douched out. If it is impossible to catheterize, I depend upon suction. If suction fails to give relief the severe pain indicating much pressure, I take down the anterior sphenoidal wall.

Acute Antrums.

A very few are of dental origin. These can be relieved only by the removal of the offending tooth. Douche the antrum and if possible, do not have the antral cavity connect with the mouth. If it does, pack the tooth socket with iodoform gauze.

The vast majority of cases are due to extension of the infection from the nasal cavity. For the pain apply electric light or diathermic heat, local depletion and suction, with the head on a horizontal plane and the affected antrum uppermost. But the best thing is to douche the sinus with saline through the normal orifice if possible, otherwise by needle puncture through the inferior meatus.

The washings from an extremely acute sinus in the very early stages will be clear to cloudy, or bloody serum. This may abort the infection from going on to a frank pus, but usually by the time the patient consults you, the antrum contains pure pus or mucopus. The antrum should be douched according to the severity of the infection every day, or every other day.

Diathermic heat is applied over the frontal or antrum, as the case may be, in the form of a small metal plate about the size of the sinus, and another larger plate on the back of the neck, passing the current up to the tolerance of the individual with the heat created, without burning the skin. This will actually raise the temperature within the sinus and, if used from five to ten minutes, will relieve the pain.

Suction is much under controversy as to its value. There should be no discussion as to its merits for diagnosis and it is often a valuable adjunct to treatment. In fact, it should be routinely used in acute infections of the sinuses where it is impossible to catheterize and douche them out. It is necessary to control the amount of vacuum used by a reliable gauge using

usually between 10 and 15 inches, or less if the patient cannot tolerate it. This should be reduced by a proper tip in one nostril, the other nostril closed and the nasopharynx shut off having the patient say k k k. The vacuum should not be continuous, but should be on for a second or two and then broken, and rapidly repeating this procedure a number of times, or in fact, as long as the suction produces pus. It is well to keep the suction down to a point below which it extravasates blood.

MEETING OF THE MIDDLE SECTION HELD IN ST. LOUIS, MISSOURI, FEBRUARY 22, 1922, UNDER THE CHAIRMANSHIP OF DR. GREENFIELD SLUDER OF ST. LOUIS.

ACTINOMYCOSIS OF THE TONGUE.

By GORDON B. NEW, M. D., and FRED A. FIGI, M. D., Rochester, Minn.

Actinomycosis is common among cattle and hogs. It occurs quite frequently in man although often it is not recognized especially around the head and neck. One hundred twenty-seven patients with actinomycosis were examined in the Mayo Clinic from January 1, 1910 to January 1, 1921. In sixty-six the disease occurred in the head and neck; in only three did it occur primarily in the tongue.

In 1826, Leblanc gave a careful description of a disease affecting the jaws of cattle, which he called "osteosarcoma." In 1845 Langenbeck first noticed the characteristic yellow granules in a case of caries of the spine in man. Bollinger (1877), however, very largely deserves the credit for the recognition of the disease as a pathologic entity. He first ascribed the peculiar enlargment of the jaws of cattle, popularly known as "lumpy jaw", to the organism that Harz had just named the "ray fungus", or "actinomyces." Israel, in 1878, recognized the organism as the cause of the disease in man. Since that time much has been written on the subject and descriptions of this condition have been recorded in practically every organ and tissue of the body.

Actinomycosis rarely occurs primarily in the tongue. It may occur in this location by metastasis or by direct extension from involved contiguous structures. Illich, in 1892, found only fifteen in 569 cases in which the growth originated in the tongue. Ruhrah, in 1899, collected 1094 cases of actinomycosis in the literature. Krymow, ten years later, was able to find only twenty-seven cases in which the tongue was primarily involved. Von Baracz reported three of this type in a series of fifty-two cases of infections in the head and neck. Schlange reports only one such case in total of sixty general infections.

Undoubtedly the tongue is more often involved than these data indicate. Three cases have been seen in the Mayo Clinic since October, 1919; none had been recorded previous to that time, and thorough microscopic examination of all specimens of inflammatory tumors of the tongue in the Mayo Clinic museum did not reveal another case. Such examination is not necessarily final since it was not possible to study the pus contained within the nodules. Four hundred thirty-seven tumors of the tongue, of all types, have been examined in the Mayo Clinic. Of these, three hundred were malignant, one hundred benign, and thirty-seven inflammatory, so that the three cases of actinomycosis of the tongue formed 0.7 per cent of the entire number.

The actinomycotic or so-called wooden-tongue of cattle is of comparatively frequent occurrence. Claus estimates the incidence of this type of lesion as 29 per cent. of all actinomycotic infections in cattle, and Leclerc as 18 per cent. Von Hollandt notes that in at least 5 per cent. of the tongues of slaughtered hogs definite, encapsulated, actinomycotic nodules can be demonstrated. The rarity of the infection of this organ in man has been explained by Poncet and Berard as owing to the fact that the tongue consists of muscle-bundles richly supplied with blood vessels and protected with a thick epithelial covering. Since the structure of this organ in animals is practically the same as in man the explanation is hardly sufficient. It seems more logical to attribute the relatively more frequent occurence in animals to the fact that their habits and mode of life bring them in more direct contact with infectious material. It is conceded that most infections due to actinomyces are of vegetable origin, such as the various grasses and grains; barley beards are supposedly the most common cause. Von Hofman and Bostroem have shown that the ray fungus is an almost constant finding on the grain beards in the tonsillar crypts of slaughtered hogs. Von Hollandt, who has conducted an excellent investigation in the slaughter houses of Dresden to ascertain the frequency of infection of the tongue in supposedly healthy hogs, states that at least 73 per cent. are due to infected plant particles.

Bostroem, who studied serial sections of primary actinomycotic tongues of cattle, was able to demonstrate foreign bodies in nearly all recent cases. The presence of such foreign bodies has also been shown in the primary lingual nodules in man by Fischer, Jurinka, Von Baracz, Harms, and Kockel. It is readily

understood how these spore-bearing particles, when thrust through the mucous membrane of the tongue, can find a favorable medium in which the anaerobic fungus may proliferate. That foreign bodies are not the only source of infection is quite evident, however, since the fungus has been demonstrated in a number of instances in the cavities of carious teeth in healthy persons, and trauma to the organ by such teeth with subsequent infection is highly probable.

In reviewing the literature we found records of thirty-five cases of primary actinomycosis of the tongue; our three cases make a total of thirty-eight. Twenty-seven of the series were in males and ten were in females; the sex was not given in one case. The disease occurs in adults; only one case has been reported in a patient under twenty, a young man of eighteen.

Incidence of the disease by decades according to ages recorded,

Age	Case
18	1
21 to 30	8
31 to 40	6
41 to 50	9
51 to 60	5
61 and over	4

The condition is more commonly found in farmers, or in persons whose rural life exposes them to infection. The disease cannot be said to be occupational since, besides farmers and their coworkers, a physician, an office girl, a janitor, a cabinet maker, two merchants, a naval pensioner, a stone mason, and a judge, are noted in the series. One of the patients seen in the clinic, a judge, mentioned a habit of chewing bits of grass while he played golf. The occupation of twenty-eight patients was recorded. Fifteen (53 per cent) were connected with rural life, thirteen (46 per cent.) were patients not connected with rural life.

The clinical history of actinomycosis of the tongue varies in different cases. The patient usually has a single, isolated nodule in the anterior half of the tongue, often near the tip, varying from 1 cm. to 1.5 cm. in diameter. This location is significant, since in giving a history the patient often mentions injury to the tongue by the teeth, or some foreign body. The condition may be acute in onset, with severe pain, throbbing, local tenderness, general malaise, and an elevation of temperature, as in abscess of the tongue. It may develop insidiously during the course

of several months, or even three or four years. Without secondary infection, the process is usually slow to develop. Enlargement of the regional lymph nodes has often been observed, but only staphylococci, never actinomyces, have been found in these, and the adenitis subsides with the clearing of the local condition of the tongue.

Diagnosis.

The diagnosis must be based on the clinical picture; and confirmed by microscopic examination. Aspiration of the contents of the suppurating nodule is usually unsatisfactory; it is best to excise the entire nodule for diagnosis. Often only a few of the characteristic granules necessary for a positive diagnosis may be present within the nodule, and unless these granules are secured in the small specimen removed for microscopic examination, it is impossible to make a diagnosis. Grossly the lesion must be distinguished from tertiary syphilis, tuberculosis, epithelioma, inflammatory cyst, and fibroma. One of our patients was referred with a diagnosis of probable malignancy. One patient was believed to have an inflammatory nodule and the third a simple infected cyst.

Pathology.

A small isolated nodule, varying in size from 0.6 cm. to 3 cm., and enclosed in a fibrous capsule, will usually be found near the dorsum, unless the entire thickness of the tongue is involved. The nodule often becomes infected secondarily with the formation of a small encapsulated abscess. This may rupture spontaneously or, in rare instances, a diffuse lingual abscess may form. Baracz's third case was of the latter type.

The overlying mucous membrane varies in appearance with the stage of the process and consequently may be normal, yellowish, or elevated and tense, as though covering a superficial cyst or a small abscess about to rupture. It rarely ulcerates. Some writers mention a peculiar bluish-red discoloration as decidedly suggestive of actinomycosis in this location, but this was not seen in our cases. The usual picture of a diffuse, indefinitely outlined induration with multiple sinuses, which depend on the ramifying granulation tissue tracts extending into the adjacent normal tissue, is rarely seen in the tongue; it was seen in only one case in the series, that of Fischer.

Most observers agree that no suppurative process is characteristic of actinomycosis, and that suppuration is always the result of secondary infection usually due to the staphylococcus.

Treatment.

The condition is treated medically and surgically. Arsenic has been used and believed to be of some therapeutic value, but its action is probably due to its systemic effect. Injections into the tongue of various antiseptics and germicides, such as bichlorid of mercury, tincture of iodin, and zinc chlorid, have been made with definite improvement, but such methods seem hazardous. The nodules have been known to disappear by the use of large doses of potassium iodid internally, (Glaisse). Poncet and Berard, however, conclude that incision or spontaneous rupture of the abscess in conjunction with the potassium iodid internally produced much more rapid and satisfactory results. Surgical treatment alone, or combined with the medical treatment, seems to be the rational procedure. Wide excision with primary suture of the wound was done in our cases, and is the treatment of choice if a discrete, isolated nodule is present. If the abscess is too large for excision, drainage with or without curetment is advisable. Daily swabbing of the wound with tincture of iodin, packing with iodoform gauze, the administration of large doses of potassium iodid, and the use of radium, as employed in the Mayo Clinic in all cases of actinomycosis of the head and neck, are unquestionably of distinct benefit.

Prognosis.

The prognosis varies with the stage of the process and the clinical picture at the time of the patient's examination. It is favorable in those cases in which a small nodule is situated well forward near the tip of the tongue; less favorable in those in which there is a diffuse area, especially if it is near the base of the tongue, or if there is a definite abscess.

Death has been reported in one case, (Mayer's), from multiple abscesses around the lower jaw, neck, and upper chest one and one-half years after excision of the primary nodule near the tip of the tongue. The abscesses were probably due to metastasis from the primary focus.

Case Reports.

Case 1 (A292538). Mrs. S., aged 63, wife of an implement dealer, came to the Clinic October 9, 1919, because of a small nodule on the tip of the tongue which had been present for two months. The nodule had been incised by her home physician one week before, and a small amount of pus evacuated.

Examination showed a small, hard, whitish nodule about 0.63 cm. in diameter on the dorsum of the tongue just back of the tip. Slight induration extended down into the tongue. The nodule was excised under local anesthesia and on microscopic examination, was shown to be actinomycosis. Fourteen months later there was no recurrence.

Case 2 (A265308). Judge F., aged 50, came to the Clinic September 17, 1920, complaining of a small nodule in his tongue. Four weeks before, he had noticed a burning sensation on the dorsum of the tongue, which he thought was due to heavy pipe smoking. Five days before coming to the Clinic, he discovered a small nodule in his tongue. He consulted two physicians who thought that it was probably malignant.

Examination revealed an indurated nodule, only slightly tender and freely movable, 1.25 cm. in diameter, just to the right of the midline of the dorsum of the tongue, and about 3.75 cm. back of the tip. The nodule was believed to be inflammatory, and, excision for definite diagnosis was recommended. Actinomycosis was diagnosed. The patient was given large doses of potassium iodid. He has not had a recurrence.

Case 3 (A339709). Mr. M., aged 46, a meat cutter, was examined in the Clinic November 3, 1920. He complained of many symptoms, most of which were apparently functional, following the nervous shock incident to the death of his son seven years before. He was referred to the Section on Laryngology, Oral and Plastic Surgery, for examination because of a small nodule on his tongue which had appeared a week or ten days before.

On examination a slightly tender, fluctuant nodule about 1.25 cm. in diameter was found on the left side of the dorsum of the tongue. The mucous membrane covering the nodule was slightly elevated and yellowish. A diagnosis of infected cyst was made, and the lesion excised under local anesthesia. Microscopic examination showed the condition to be actinomycosis. The patient was given large doses of potassium iodid.

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X-RAY DIAGNOSIS OF UNUSUAL LARYNGO-TRACHEAL AND ESOPHAGEAL CONDITIONS AND DISEASES.

By SAMUEL IGLAUER, M. D., Cincinnati, Ohio.

With perfection in the technique of esophagoscopy, there may be a tendency to overlook the value and importance of the X-ray diagnosis of esophageal lesions. The information obtained by the latter method is often a great aid to the esophagoscopist, and is of great value first in determining the functional activity of the esophagus with the fluoroscope, second in getting the static condition of the esophageal lesion and finally as an aid to the introduction and fixation of appliances within the lumen of the esophagus or larynx.

Careful X-ray study of the esophagus will often reveal some unexpected and often astounding conditions. I should like to cite a series of cases to illustrate these unusual findings:

Case Reports.

Open Safety Pin, Pointing Downward. The first case is that of a safety pin in the esophagus of an eight months old infant. While "changing" the child the mother, a physician's wife, missed the pin at 1 o'clock in the morning, and told her husband that the child had swallowed the pin. He was inclined to doubt the occurrence of the accident, but the child was brought to the city and X-rayed by Dr. Doughty, who was much surprised to find the pin with the point down, lodged in the lower third of the esophagus. At 6 P. M. of the same day, upon introducing the esophagoscope, curds of milk were found in the lower esophagus. Upon withdrawing the tube the patient regurgitated the curds and the pin, which fell upon the floor. In this case the X-ray showed the pin pointed downward, in what is supposed to be an impossible position. Two explanations of this condition have been offered. One is that the pin may be swallowed closed and may open in the esophagus, and the other is that the pin may reach the stomach open and in the subsequent act of vomiting, may lodge in the esophagus with the point downward. It is difficult to conceive that either of these explanations is more simple than that a patient may occasionally swallow a pin with the point downward. In this particular case the parents are certain that the pin was open when swallowed and that there was no vomiting after the pin had been swallowed. In the case cited above the pin was one inch long, and had a sharp point, which one would naturally believe would stick in the mucosa. Should a similar case occur with the same X-ray findings, one might consider the advisability of giving the patient an emetic and thus recover the pin without resorting to esophagoscopy.

Non Opaque (X-ray) Foreign Bodies in the Esophagus. The esophagus may be partially obstructed by a substance translucent to the X-ray. In such cases the diagnosis of the presence of a foreign body is often attended with some difficulty. I should like to cite three cases illustrating this subject.

Case 2. Mrs. B., age 58, gives the interesting history that when she was a child she was found upon the ground, unconscious, with a pool of blood issuing from her mouth and a dead lizard lying in the blood. Her physician and family allowed her to grow up with the belief that she had been bitten by the lizard, which caused a hemorrhage. She gives a history of having had a persistant purulent expectoration all her life until within the last few months. She stated that she frequently had some difficulty in swallowing, but that she usually managed to get the food either down or up. Her present trouble occurred two days before I saw her. While eating some ham she suddenly was unable to swallow any more food. X-ray examination of the esophagus by Dr. Doughty, was as follows:

"There is a rather definite obstruction opposite the seventh dorsal vertebra. This obstruction shows a slight dilatation at the immediate point, but there is shown no definite dilatation from there up. The characteristic marked thinning, just below, usually seen in these obstructive cases due to a malignant type of growth, is not shown.

In the stereoscopic chest films, the entire left side is shown to be slightly fibrous. The heart and mediastinal shadows are shown to be to the left, as is frequently seen in these old fibrous types.

Below the point of obstruction, there can be seen about the normal type of lumen of the esophagus, as far as can be made out.

With the history of this case, it would appear to me that there is some limitation in dilating the esophagus throughout its course, possibly, slightly more at this point. In eating, the food as described by the patient, a few days ago, may have blocked at this point—that is, a rather large portion of the solid food—, and which has not, as yet let go and which does not pass on down; and therefore causes the obstruction. At the seventh dorsal vertebra, there is shown this slightly increased dilated area, which gives this impression".

The diagnosis then was a non-opaque foreign body obstructing the esophagus. I made an additional diagnosis of angulation of the esophagus by the traction of adhesions pulling it toward the left and presenting a very unusual picture. (Dr. P. Armand-Delille, Ref. J. A. M. A., Feb. 11, 1922, has pointed out that "in ten of 300 patients with chronic tuberculosis processes in the lungs, the shriveling of tissue and other causes had pulled or pushed the trachea so far to one side that it was liable to be mistaken for a cavity"). Owing to the angulation of the esophagus I anticipated some difficulty in introducing the esophagoscope, but this did not prove to be the case, and I removed two pieces of partially ossified bone from the obstructed point in the esophagus, which was found to be slightly ulcerated at this point. Without an X-ray examination the peculiar distortion and narrowing of the esophagus could not have been determined in advance.

Case 3. The third case was that of Mr. H. L., age 25, who three days before, while eating a veal chop and potatoes had a severe choking spell

with marked dysphagia since the accident, and inability to swallow anything but semi-fluids. Dr. Lange reports that: "Fluoroscopic observation of the act of swallowing revealed no obstruction to the downward passage of barium suspension but large opaque capsules lodged in the esophagus about on a level with the first dorsal vertebra. The film shows three capsules lodged above a *transparent obstruction* which is *partly outlined by a coating of barium left from the barium suspension. It seems that the watery suspension of barium passes the obstruction freely but the capsules do not.*" In this manner the site and to considerable extent the size of the foreign body, which proved to be a large piece of meat could be clearly determined.

Case 4, seen in consultation with Dr. A. Friedlander, is even more interesting. A boy, aged 6, swallowed lye two years before we saw him. Since the lye-accident he has had more or less difficulty in swallowing. Seventeen days before he was brought to us, he told his mother that he had swallowed an orange seed. Since that time he had been unable to retain any food, not even water, and vomited almost immediately after eating. He had lost about 12 pounds. X-ray examination by Dr. S. Brown showed that opposite the 9th rib posteriorly there was a marked stenosis of the esophagus with a sausage like dilatation above, and with the barium trickling through in a very thin stream below this point. Upon close scrutiny it appeared to me that there was a filling defect in the esophagus at the point of constriction, which I venture to suggest might be due to the presence of an orange seed. The diagnosis then was "*Stricture of the esophagus, obstructed by a foreign body, with dilatation above the point of constriction.*" The foreign body was easily located with the esophagoscope and proved to be an orange seed. X-ray examination made a few days after the foreign body had been removed, much to our surprise showed that the esophageal pouch above the stricture had disappeared, so that by the X-ray examination we were able to state that the patient had *had an acute dilatation of the esophagus,* (comparable to an acute dilatation of the heart) which had disappeared after the obstruction had been relieved.

Cases 2 and 4 would indicate *that the presence of a filling defect in a stenosed esophagus may be due to the presence of a foreign body.*

Unexpected X-ray Findings. *Case* 5. The X-ray examination in this case proved to be of great value to both the patient and the physicians. Mrs. S., aged 79, was seen at her residence, in consultation with Dr. List. She stated that she had been having increasing difficulty in swallowing for the last three years, and that some of the food would occasionally regurgitate and that she had lost about 17 pounds. She was bed-ridden for about a week before I saw her. Owing to her extreme age, the natural inference was that she was suffering with a progressing carcinoma of the esophagus and it seemed scarcely worth while to subject her to any examination. Nevertheless, we sent her to an X-ray laboratory, and upon swallowing a barium suspension a large diverticulum of the upper end of the esophagus was discovered. (Dr. Schroeder and later Dr. Brown) In order to rule out malignant disease, the esophagoscope was passed into the pouch but no carcinoma could be demonstrated. Gastrostomy was then performed by Dr. Ransohoff. Since that

operation the patient has gained considerably in weight and may live for many years as a result of the X-ray examination and the operation. (Note.*)

An interesting accident occurred with this case. Immediately after the esophagoscopy, during which some cocain was used, the patient was again given barium to swallow, and the X-ray picture showed that some of the barium had entered and partially outlined the lower bronchial tree in both lungs. However, the patient expectorated all of this barium suspension, without any damage to the lung structure. This is in line with the experimental insufflation of the bronchi with bismuth, as described in Jackson.

Case 6. The following case shows how the X-ray may occasionally make a false diagnosis, and proves *that things are not always what they seem.* A child was brought to the hospital with a statement that it had swallowed a 5-cent piece. X-ray examination showed the foreign body in the usual position at the upper end of the esophagus. Upon introducing the esophagoscope, I informed the interne that the patient had 6 cents in the esophagus, for I could make out the edges of two superimposed coins, one brass and the other nickel. The coins were withdrawn together and proved to be a dime and a penny, so that I was 5 cents to the good, despite the mistake in the diagnosis.

In cases of cardiospasm, it seems very likely that by inducing pneumoperitoneum and taking an X-ray picture of the diaphragm we may occasionally find pathological adhesions of the liver, encroaching upon the sub-diaphragmatic portion of the esophagus and giving rise to the symptom complex cardiospasm. This is well illustrated in a case previously reported. (New York Med. 1922.) In this case, the X-ray films show an enormously dilated esophagus with adhesions of the liver to the diaphragm in the area corresponding to the subdiaphragmatic portion of the esophagus.

Note. *(Since the above was written the pouch was successfully removed by Dr. Ransohoff.)

MEETING OF SOUTHERN SECTION, HELD IN BALTIMORE, MARYLAND, FEBRUARY 4, 1922, UNDER THE CHAIRMANSHIP OF DR. T. CHEW WORTHINGTON OF BALTIMORE.

ITEMS OF PROGRESS FROM LAST YEAR'S LITERATURE OF OTORHINOLARYNGOLOGY.

By H. O. REIK, M. D., Baltimore, Md.

When your Chairman invited me to participate in this meeting I accepted the invitation more for the reason that it would force me to do a thing I knew I should want to do, but might otherwise find an excuse for not doing, that is, to foregather once again with this group of old friends and fellow workers, rather than because I had anything special to talk about. You are all aware of the fact that I have been out of active practice for the past five years and that I can no longer contribute to medical science out of my personal experiences, but it is possible that I may add something to the gayety of nations if I cannot increase the sum total of your store of knowledge.

During the past year, while laboring for the establishment of a unique publication, the International Medical and Surgical Survey, it has been my privilege to sit in the Conning Tower, as it were, and to observe with a more or less critical eye the literary product of the vast horde of medical scientists at work all over the world. I assure you it is an interesting position to occupy and would be most enjoyable if one had the time to calmly study and ponder over the many valuable contributions encountered, instead of having to work overtime trying to put this mass of material into assimilable form for the rest of you to digest. The range of things possible for me to write about being, then, something told of the experiences of others, and the range of subjects in which you would be most interested being limited to the scope of our specialty, it occurred to me to look over the abstracts of the otorhinolaryngologic literature of the past year and to select for discussion a few of the most important items therein considered.

You are perhaps all familiar with the fact that there are in the world 26 first-class periodicals devoted solely to otorhinolaryngology; these appearing in six different languages—English,

French, German, Italian, Scandinavian and Spanish. For this portion of Section 8 of the Survey abstracts are made of every original article in this group of periodicals and to this is added all abstracts relating to this specialty that appear in the more than 400 other leading general medical journals of the world. From such sources we obtained and published during 1921 the abstracts of 1039 original articles concerning this special branch of medicine. It is not an easy matter to decide which, from all of those papers, was the most important contribution to medical knowledge, even after waiving aside the question of the relative value of scientific investigations and clinical observations. The few that I shall speak of have been selected because of their supposed practical bearing upon the day's work and I trust may prove to be of interest to you.

Quite naturally, the tonsils have held the center of the stage. I question the wisdom of starting a discussion upon the technic of tonsillectomy and so will pass over that problem with but a single reference, a paper by Coakley describing a new, simple and practical method for ligating vessels in the tonsillar fossa, and will then review, in this connection, three groups of papers relating to (1) the radiologic treatment of tonsils, (2) the performance of tonsillectomy in the treatment of quinsy, and (3) the occurence of pulmonary abscess following tonsil operation.

Coakley, in a recent paper urging the same surgical care of bleeding vessels during and after tonsillectomy that is employed by surgeons operating on other parts of the body, describes his own technic of tonsillectomy, with which you are all familiar, and presents the method of ligation that appears to be simple and effective. He uses the Allis hemostat with four mouse teeth; the handles of this instrument being made 7 inches long. A straight hemostat is used for picking up vessels on the posterior wall of the fossa, whereas a slightly curved one is more useful for seizing vessels in the supratonsillar region or to displace the pillars so as to reach bleeding points at the lower pole. After much experimenting, small black silk braided fishline, which has a tensile strength of 8 to 14 pounds has been found to make the best ligature. A piece of this silk, 14 inches long, sterilized by boiling, has a slip knot tied in the middle of it, the loop being large enough to pass over the handle of the hemostat holding the vessel. The knot should be drawn sufficiently tight to let the free part slide with slight friction. The non-slip par:

of the loop is grasped close up to the knot with the end of a pair of slightly curved, long handle forceps. The loop is placed over the hemostat and carried down the shank and over the end of the hemostat so as to engage the bleeding point. During this manipulation the loop is gradually lessened in size by pulling lightly upon the sliding part. The placing of the loop is done entirely with the forceps until such time as the loop is over the end of the hemostat, when the free end is drawn tight and the vessel is ligated. The hemostat and clamp are then removed and the free ends of the ligature picked up and cut off close to the knot; although this knot is the famous slip knot, when pulled tightly it cannot slip and the ligature may be found in the fossa from six to twelve days later. If several forceps are threaded with such a slip knot ligature at the beginning of the operation, ligation may be performed as quickly as, if not more quickly than, a surgeon would ordinarily tie a vessel in an open wound.

There have been during the year a sufficient number of papers on the treatment of diseased tonsils by radium or X-rays to justify our serious consideration of that method of treatment. The scientific basis for an attempt to induce atrophy of hypertrophied tonsils by the Roentgen rays is the established fact that this element will destroy lymphoid tissue in other parts of the body. The technic has been thoroughly described by Murphy, Witherbee, Craig, Hussey, and Sturm, and their reports of results are fairly and conservatively set forth: Atrophy did take place in the majority of instances, the crypts opened and drained in most cases and such organisms as the streptococcus hemolyticus disappeared; this last fact being attributed to the effect of drainage and not to any bactericidal effect of X-rays. These writers have not offered the treatment as a substitute for tonsillectomy, but suggest its applicability in two directions: first, for determining what tonsils are the focus of systemic infections, and secondly, as a means of relief where surgical intervention is contra-indicated. In support of this work one finds an interesting observation by Byron Sprague Price, who states that while intentionally X-raying a group of patients for cervical adenitis and only incidentally raying their adenoids and tonsils, he observed that a considerable number of them showed improvement in their tonsillar and adenoidal conditions sufficiently marked to permit of their escaping operations. This led him to submit 23 cases of diseased tonsils, 14 of them having

accompanying adenoids, to X-ray treatment, with the result that in every case the adenoids practically disappeared and the tonsils took on the appearence of health.

Concerning the value of radium, instead of Roentgen rays, we find two excellent papers by Francis H. Williams and Walter Wells, respectively, both of whom claim that radium possesses the advantage that it can be applied directly to the seat of trouble instead of being obliged to pass through healthy tissue on its way to the diseased area, and both of whom report the same favorable results as are set forth by the roentgenologists. A very fair summary of the present status of this question has been presented by Osgood of Boston, who says, in brief, that radiation for diseased tonsils is not to displace tonsillectomy; that it is a method which can produce atrophy without hemorrhage, discomfort, or loss of time; that it does not sterilize the tissues, but improves the condition of the crypts through mechanical drainage; that it is a new method which has given gratifying results clinically, but has not been sufficiently tested; and, that it has dangers that must be carefully considered.

Advocacy of tonsillectomy as the proper treatment for peritonsillar abscess has been the theme of three important papers, all arguing that the proper treatment for quinsy is the immediate enucleation of the tonsil regardless of the stage of peritonsillar inflammation. The points of advantage claimed are the more prompt cure of existing local condition, the abortion of systemic infection, and the prevention of recurrences. That the method is not without danger is illustrated by the fact that Heller reports 48 cases so treated, with 2 deaths. In these 2 fatal cases high temperatures and evidences of severe sepsis followed immediately upon the operation; one death is explained on the supposition of lymphatic leukemia, and the other by status lymphaticus.

Those who have recently written upon the subject of lung abscess following tonsillectomy have given consideration principally to the manner of production of this postoperative complication. The consensus of opinion seems to be that in the majority of instances the pulmonary infection results from aspiration of infective secretions or debris, while under the influence of general anesthesia; 2 cases have been reported, however, in which lung abscesses developed after tonsillectomies done under a local anesthetic. Voorhees calls attention to the somewhat careless manner in which cheesy masses are squeezed

out of tonsil crypts into the pharynx and the ease with which they might infect the respiratory tract. The possibility of the blood stream conveying to the lungs infective material from a thrombus in some vessel of the tonsillar fossa has been considered as an important factor in some cases. The only practical suggestions offered for the avoidance of this serious complication refer to measures for keeping the throat free from secretions during and after the operation by careful sponging or by the use of the vacuum suction apparatus; as one means of accomplishing this, Clifford Walker has devised a suction dissector which consists of a hollow tube whose end is flattened like a fish tail.

The subject receiving the greatest consideration, if one may judge by the number of papers devoted to it, has been the question of radiologic treatment of malignant affections of the throat and nose, including the accessory sinuses. Crosby Greene has furnished the best summary of conclusions in these statements: (1) In malignant disease of the regions considered, the chief value of radium lies in its supplementary rather than primary worth and it should not supplant operation. (2) In most cases palliative results in the way of retardation of growth and relief of pain may be expected. To which may be added the suggestion of Freudenthal and others, that pre-operative applications may be advisable in certain cases to lessen the vitality of cancer cells and limit the possibility of metastasis. Amersbach, in an article in the Deutsche Medizinische Wochenschrift makes a statement that so far the principal effects of radium treatment have been arrest of pain in the inoperable cases, and as an adjunct to surgical treatment, and, that no cures have yet been proved as attributable to radium alone. Didier, in the Journal des science Medicale de Lille presents an interesting résumé of this subject, with an attempt to compare the effects of radium and x-rays on the different conditions presenting. As various types of neoplasms have different degrees of susceptibility to radiation, it is very important to determine beforehand the nature of the lesion to be treated. Roentgen rays are not as powerful in action, but their field of action is much more extensive than that of radium. In sarcomas the x-rays should be used; in epitheliomas, radium gives the best results. If the accessory nasal cavities are involved, X-rays give good results in sarcoma, but have been found unsatisfactory in epithelioma. In the mucosa of the pharynx the majority of tumors are

epitheliomas or lymphadenomas and X-rays give the best results. Radium has been successfully used in tonsillar tumors. Intralaryngeal tumors must be treated surgically and, after this, radium applied as indicated. As a general rule, X-ray therapy is less dangerous than radium therapy, but it is chiefly palliative.

Ozena was accorded a surprisingly large number of papers during the year; 17, I think. Though there is still much discussion as to the etiology of this condition, evidence seems to be accumulating to prove that the Perez bacillus is the causative factor. Hofer claims, and is supported by several other German writers, that vaccine therapy produces 8 to 10% of cures, 15 to 20% of failures and marked improvement in all other cases. The use of scarlet-red in 4% solution continues to receive an occasional favorable report and one author has found it most serviceable when the emulsion is made from quince seeds, the viscid character of which causes a close adherence to the mucous membrane in spite of secretions. Two Italian confreres, Bruzzi and Podesta, have called attention to the possible use of carbohydrates in fighting this disease and have experimented with glucose, saccharose and lactose. The fetor that accompanies this condition arises from a decomposition of protein substances by bacterial fermentation, with the formation of sulphur compounds, and the growth of the Perez bacillus is fostered by this decomposition. In the normal nasal cavity there is an abundant bacterial flora possessed of glycolytic powers. The scope of this treatment is to arouse the activity of such micro-organisms to the fermentation of sugar and production of acids which are detrimental to the Perez bacillus. The method of treatment consists in thorough cleansing of the nose by irrigation with alkaline solution and the application of cotton tampons soaked in glucose solution; these to be changed three or four times a day at first, and daily spraying substituted for the tamponage as improvement progresses. The solution used consisted of saccharose, 15 gm. to distilled water, 15 c.c.; diluted honey was of equal efficacy. Very satisfactory results are claimed for the method.

One might also have observed in a glance at recent literature a very active renewal of interest in West's operation for the relief of dacryocystitis, or at least in some method for extirpation of the lacrimal sac. The Totti operation of attacking the sac from the external aspect and the Mosher modification thereof have their strong advocates, but the intranasal procedure is

still preferred by the majority of operators. It would seem that nearly everybody attempting the operation has suggested some slight modification of it, but it is questionable if anyone has improved upon the technic devised by West himself. It may be interesting to remind you that West was a Baltimore boy and those of you who remember him must be pleased to observe that he has made his way both in practice and in scientific special work in the very center of German rhinolaryngology—in Berlin.

A somewhat interesting piece of work is reported from Naples by Azzo Azzi (Riforma Medica) with relation to the effect of heat and cold applied to the skin on the temperature of the mucous membrane of the trachea and bronchi. Making observations upon animals and on men upon whom tracheotomy had been performed, it was shown that ice or currents of cold air applied to the skin cause a fall of from .4 to .9 degree in the trachea, even when there is no fall in the general body temperature. This helps to explain the development of respiratory diseases from chilling of the body surface and also the therapeutic effect applied to the thorax for the control of hemoptysis; the ice does cause a vasoconstriction in the lungs, with consequent arrest of hemorrhage. In this connection it will not seem out of place to consider other measures for the use of heat and light therapeutically. Čemach, in the Monatsschrift für Ohrenheilkunde und Laryngo-Rhinologie urges the use of red hot light for the arrest of inflammations in the early stage by inducing resorption; incidentally, analgesia results almost immediately upon the first application. The method is not without value in more advanced inflammatory conditions, but when the tissues are so damaged that immediate regeneration is impossible, the best that can be hoped for is the hastening of suppuration and pointing. Any electric lamp of 300 to 600 candle power may be used and should be held at a distance of 10 to 12 cm. from the affected part; applications varying from 30 minutes to an hour as the patient gradually becomes accustomed to the heat. In inflammations of the accessory sinuses, tonsils, and mastoid, the treatment may be employed morning and evening, care being taken in working about the face to avoid burning the eyes. It is, of course, but a new method of employing a very old therapeutic agent—heat.

Diathermy, or the coagulation of tissues by heat generated in the tissues themselves by the passage of high frequency currents of considerable strength, is a comparatively recent innovation

which has been received very favorably. It seems to be of special value in the treatment of growths, whether benign or malignant, and of diseased tonsils where enucleation is contra-indicated. Currents of from 300 milliamperes to 2 amperes have been used. A large negative electrode, thickly padded and thoroughly wet with saline, is placed on the back or chest; the growth is dried, a small active electrode applied and the current gradually increased up to the desired strength. Passing a strong current through a small electrode generates great heat in the underlying tissues and these coagulate and slough away. The heat sterilizes the tissues, seals the blood-vessels and lymphatics and prevents septic absorption. In operations about the larynx edema may appear and therefore tracheotomy should always precede diathermy in this region. The advantages claimed for the method are: absence of shock; pain less than by surgical measures; diminished danger of metastasis or of sepsis; a bloodless operation that may be repeated if necessary; and rapid convalescence. The only disadvantages seem to be that healthy as well as diseased tissues may be burned and the possibility of injuring important vessels or nerves that cannot be seen. The only published reports so far have come from our English brothers, but W. J. Harrison and Sir William Milligan are conservatively enthusiastic about it.

I should like to have had time to discuss a number of very interesting articles covering anatomical research, especially those relating to the labyrinth and to the histology of deaf mutism, but I fear that I have already occupied too much of your time. In the field of otology, it would have been my pleasure to praise the excellent work of our ex-President, Dr. Dean, in developing his audiometer and, in that connection, to have called attention to the several inventions of Griessmann published in the German journal of Anatomy, Physiology, Pathology and Therapeutics. His "Autodion" is apparently not unlike the audiometer in principle; his "Otosklerometer" makes possible a quantitative determination of the motility of the ossicles; and, his "Ototelegraphone" is said to supply a uniform hearing test which speech cannot give because it generates a normal uniform speaking tone without acoustic distortion and with much clearer transmission than by an ordinary telephone. Also, your attention might have been called to the "Vactuphone" recently described by Hanson in the Volta Review as a vacuum tube telephone which, as a hearing aid for the deaf, is said to

convey and reproduce human speech more clearly and distinctly than any electric hearing device previously produced.

In closing, permit me to bring to your attention the reports of the American Association of the Hard of Hearing, which organization has grown out of the indefatigable labors of one of the founders of this Society, Wendell Phillips, who started the New York League for the Hard of Hearing, and who for years has been engaged in these philanthropic efforts to benefit suffering humanity.

The enlightened physician recognizes the fact that he has not fully acquitted his obligation when he has done all that can be done through medicine and surgery for relief of an incurable affection; he still owes that patient consideration and advice as to the means for making the best of his bad condition. There are literally thousands of deaf persons whose lives can be made happier by proper guidance; by advice that they should receive from their special medical counsellor, rather than through lay channels. They should be taught the importance to them of learning lip reading and the importance of maintaining social intercourse like normal hearing people. There is need for a league for the hard of hearing in every community large enough to support an otologist, and who but the otologist should take the initiative in starting such a beneficial work?

A METHOD OF DEMONSTRATING THE SURGICAL ANATOMY OF THE MASTOID.

By JESSE WRIGHT DOWNEY, JR., M. D., Baltimore, Md.

Variations in the normal anatomy of the temporal bone are so frequent, and minute details that might seem of academic interest only, are of such vast practical importance; that the aural surgeon must constantly visualize a more or less composite anatomical picture that shall include, not only the external aspect of the mastoid region, but more especially the complicated system of cells that exist within the mastoid and petrous portions and which are, as Sir Charles Ballance so clearly emphasizes; "quite distinct from the medullary, the diploetic and the Haversian spaces proper to the bone substance".

Of course, it is most essential that one first acquire his mental impressions of the anatomy of the mastoid and tympanic cavities in their relationship one to the other and to contiguous structures by the study of many temporal bones; and by the practice of the various surgical procedures upon the cadaver. Knowledge so gained may be, however, of only relative value in the performance of the actual mastoid operation for the cure of disease, for the most constant concomitant of a pathological process is the change wrought in the appearance, the structure and the relationship of the part involved and; therefore, it is also necessary to be prepared to meet anatomical conditions which are the result of the disease itself.

It is these variations in both normal and morbid anatomy which place the modern mastoid operation in a class of surgery requiring not only technical training but the ability to interpret operative findings that follow the exception rather than the rule; and as it is by no means easy to describe one's findings at the time of operation either in written words or by flat drawings, a clinical history, especially when presented before a class of students, or a medical society, can be much enhanced by a model of the operative findings. No operation lends itself better to modeling than does the mastoid operation and I hope to demonstrate that the method is practical.

The twelve models which I wish to show have been modeled in artist's water-clay from actual operations. Measurements were made at the time of operation, dictated to a nurse and the model made within a few hours after the operation. Any otologist will find the process surprisingly easy, if he will take a piece of clay and imagine he is operating. The attempt to reproduce the appearance of the mastoid cavity teaches one to pay the strictest attention to details at the time of operatio· and certainly good mastoid surgery, like genius, is dependent upon the ability to take infinite pains.

Twelve models of actual operations were exhibited, demonstrating:—Extensive destruction of the mastoid process. Anomalous positions of the lateral sinus. Zygomatic cells simulating the antrum. The difficulty of reaching the antrum in certain sclerotic bones. The marked alteration in anatomy occasioned by chronic suppurative conditions, etc. Stereoscopic and simple photographs of these models are being prepared and will be published later.

SUBCUTANEOUS EMPHYSEMA OF THE NECK AND CHEST FOLLOWING TONSILLECTOMY IN AN EPILEPTIC. RECOVERY.

By SYLVAN ROSENHEIM, M. D., Baltimore, Md.

Osler defines emphysema as the condition in which the infundibular passages and the alveoli are dilated and the alveolar walls atrophied. Surgical emphysema, subcutaneous or traumatic emphysema is that condition where air or other gases are found in the subcutaneous tissues.

Among the varieties of subcutaneous emphysema are: Inflammatory or gangrenous emphysema—caused by gas bacillus of Welch. Congenital emphysema—occurs in infants where there is a congenital weakness of the lung. A spell of violent coughing may cause it. Spontaneous emphysema — may occur after extensive extravasation of blood. It is due to the gas produced by acids such as lactic, acting on the contained blood. Traumatic emphysema—This may be of external origin such as wounds of the face, stab wounds of the neck or chest, etc. or of internal origin from the nose, pharynx, larynx, trachea and lungs, wounds or disease of the gastro-intestinal tract or following excessive straining during pregnancy, etc.

There are two ways in which the air may get into the subcutaneous tissues from the lungs:

1. The intrapleural route. The air must pass through the visceral and parietal pleura. In order to do so, adhesions must exist between the pleurae at this point.

2. The extrapleural route. Air sacs on the periphery of the lung rupture and the air follows the line of least resistence travels beneath the visceral pleura to the hilum of the lungs, follows the reflection of the pleura on the big vessel and enters the mediastinum and then follows the fascial planes into the tissues. There is in these cases an increased intrapulmonary pressure.

Subcutaneous emphysema following removal of the tonsils is apparently, from a survey of the literature, such a rare occurence that the following report should be of interest.

Case Report.

S. B., male, aet. 21 yrs., entered the Hebrew Hospital March 11, 1912 under the care of Dr. Irving J. Spear for thorough examination and treatment for epilepsy.

The family history is unimportant. The patient has had measles, chicken pox and mumps. He gives a history of epilepsy, the attacks having occurred at irregular intervals since the age of 14 years.

At the present time he complains of some trouble in hearing. Besides the presence of hypertrophied tonsils and adenoids the physical examination was of no special interest. The tonsils were removed under ether anesthesia. A weak solution of adrenalin chloride was first injected around the tonsils and then the tonsils removed by sharp dissection with the knife. It was not necessary to tie any vessels. The adenoids were then removed by curettement. There was considerable trouble during the operation, due to the patient taking a poor anesthetic. When partly asleep he had a slight epileptic attack. The operation lasted one hour, having began at 3 P. M. That evening the patient's condition was not good. He breathed very heavily and frothed at the mouth expectorating some blood clots. He seemed to struggle for air. His temperature was 103.2°, pulse 150. He was not seen at this time by the writer. The next morning an emphysematous swelling was noted in the neck on both sides extending downwards almost to the clavicle. The breathing was easy and the patient was more comfortable but complained of pain in the neck and abdomen. The stools contained some blood. The leucocyte count was 37,000. The next day the emphysema had extended farther downwards, but the patient's condition was better. The leucocyte count was 26,000. The hemoglobin was 85%. During the next two days the emphysema extended, covering the front of the chest and the axillary region. His general condition commenced to improve. His temperature returned to normal on the tenth day and the emphysema gradually receded. During his stay in the hospital he received hydro and electrotherapeutic treatment as well as medicinal. He had but one epileptic attack while in the hospital. He was discharged March 20 much improved.

We have been able to find but one case similar to ours in the literature of this subject. That was reported by Parrish in the Laryngoscope in 1910. It occurred in an adult male following the removal of the tonsils with tonsillotome and punch under ether anesthesia. There was rather free bleeding during the operation and a button hole was made in the posterior pillar. Immediately following the operation there was extensive emphysema of the neck and face and anteriorly on the chest. The head and neck were extended, the breathing was rapid and shallow, the pulse 128 and thready. By retaining the mouth open with a gag and flexing the head the condition of the patient was immediately improved. Nothing abnormal was seen in the

nose or throat. The air gradually was absorbed first from the face and neck and later from the chest and there was a complete recovery.

Woodward reports a case of emphysema of the head and neck following the perforation of the anterior pillar of the fauces by a pipe stem. Stewart had a case in which there was an extravasation of blood into the subcutaneous tissues of the neck following the removal of the tonsils in a boy aged seven years. This case ended fatally. The prognosis, of course, depends upon the cause. The cases following influenza and pneumonia usually end fatally. The prognosis is in most of the cases good. In our case although there was apparently some infection as indicated by the elevation of temperature and the leucocytosis, the patient appeared at no time very sick. There is no special treatment required for non-inflammatory cases. When very extensive some have recommended multiple punctures.

What is to be learned from this case? When one considers the local and general complications that may follow tonsillectomy such as mutilations of the palate, hemorrhage, local infections of the wound, general sepsis, infections of distant parts as the serous cavities and lungs, it seems that the lesson is quite clear. It is to perform the operation so as to conform to all the rules and regulations of modern surgery. Reading from week to week of the various new instruments devised to remove the faucial tonsil. one wonders, if we of this specialty, do not pay too much attention to the instrumentation, and not enough to the principles involved. According to our ideas, developed through twenty-one years of work in this field, we have come to the following conclusions:

1. That a careful preliminary examination of each patient should be made.

2. When the operation is performed under general anesthesia. ether is the anesthetic of choice. This should be administered only by a specialist in this line, using the warmed ether vapour administered through a tube inserted on the top of the tongue depressor as in the Davis mouth gag. The operating table should be at an angle of about 25 degrees with the horizontal. the head of course being down, thus preventing aspiration of blood and debris into the trachea and lungs. The precautions will we think avoid the complications of ether pneumonia and lung infections. In adults we prefer the use of local anesthesia.

thus avoiding any possible complications from the use of ether as well as the unpleasant ether sickness. The main point in the use of the local anesthetic is to use a very weak solution for injection and allow none to be swallowed by the patient. Usually a hypodermic injection of morphia and atropine is given one half hour before the operation. I use an eight per cent solution cocain with four drops of 1-1000 adrenalin chloride solution to the ounce, previously swabbing the throat with a moistened applicator on which powdered cocain is spread. I have been using this method for seventeen years with as yet no untoward results.

3. Hemorrhage, long the bugbear of this operation has been reduced to a minimum by the procedure to be described. However, when it does occur, pressure is made by one or more sponges held in Kelly clamps, placed in the tonsillar fossae and if this does not stop the bleeding, the bleeding point or vessel is grasped with forceps and tied with catgut. We have never found it necessary to use the vacuum apparatus to keep the throat clean, the slight bleeding being readily taken care of by sponges held on Kelly clamps. Where there is a suspicious history of bleeding previously it might be well to apply a solution of cephalin 10% to the fossae after removing the tonsils.

4. I am fully persuaded that all operative procedures, based on the guillotine principles are reversions to crude tonsillotomy performed years ago. The operation is performed as follows: In ether cases, the space between the capsule of the tonsil and the fascia of the superior constrictor is infiltrated with normal saline solution to which four drops of 1-1000 solution of adrenaline chloride to each ounce is added. The tonsil is grasped at the upper pole with tonsil forceps and a small incision is made in the plica triangularis. The flat blade of the knife is inserted in this opening and the dissection of the upper lobe from the supratonsillar fossa is effected, an assistant meanwhile assisting the operator by following the dissecting with a broad retractor. The blunt dissection is continued with a small hard gauze sponge held in a Kelly clamp. When the tonsil is dissected free from the pillars, the operation is completed with the Pierce-Mueller snare. Any bleeding that occurs is controlled as outlined above.

If adenoids are present they are then removed and the adenoid bleeding is controlled by a sponge made to fit the nasopharynx. While this pack remains in the nasopharynx a few minutes, the

tonsillar fossae are inspected for bleeding points. The time consumed for the operation averages from fifteen to twenty minutes under ether. When cocain is used the time consumed is usually the same, being dependent mainly upon the idiosyncrasies of the patient.

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ADAM POLITZER—IN MEMORIAM.

By HARRY FRIEDENWALD, M. D., Baltimore, Md. *

Adam Politzer died in August 1920, at the age of 85. His relation to modern Otology was unique; his name is associated with Otology from 1861 to 1920, a term of almost 60 years, the period embracing modern Otology; his scientific contributions were fundamental and of the highest importance; he was the teacher to whom thousands went for instruction and inspiration. No wonder then that we regard him as one of the great founders of the science of modern Otology.

Graduating in 1859, he began immediately to devote himself to otologic problems, working under the leading physiologists and anatomists of the day, such men as Ludwig of Vienna, Kölliker Heinrich Müller, Helmholtz, Claude Bernard, Rudolf König and studying clinical otology under Tröltsch, Toynbee and Meniére. In December, 1861, he began his career as private dozent in Otology, the first in the University of Vienna; in 1870, he became professor extraordinarius; in 1896, professor ordinarius and he taught until 1907, when having reached the age limit of 72 according to the Austrian law, he retired from the University, where he had taught for almost half a century. On this occasion he was presented by his former pupils with a handsome relief portrait in bronze.

His work in the anatomy and pathology of the ear is extensive and most important. I need but remind you of his "Anatomische and Histologische Zergliederung des Gehörorgans" in 1889. His anatomical and microscopical preparations are well known and bear alike the marks of the scientist and the artist, and they form next to that of Toynbee, the richest collection extant.

Among the conditions that were first recognized and described by Politzer are otosclerosis, panotitis, otitic changes in leukemia; he added greatly to our knowledge of the labyrinthine lesions of the deafmute, of purulent otitis interna, etc. The simple method of inflation with which his name will ever be associated was introduced by him in 1862.

*The speaker showed colored drawings, and plaster models made by Prof. Politzer, microscopic sections prepared by him, a collection of his published works and other personal mementos.

There is no greater service to otology than that rendered by Politzer in the diagnosis of aural conditions through his artistic and lifelike colored drawings of the drumhead in various conditions of disease. His first publication was his "Beleuchtungsbilder" in 1865, republished and enlarged in many subsequent editions and still one of the most popular of all aural atlases.

Politzer's Lehrbuch, first published in 1878 reappeared in many editions and translations and has for a generation held its own as the leading textbook and work of reference.

His marvellous gifts as a teacher are indicated by his popularity; 7000 foreign students alone, came from every civilized country in the world to seek his instruction; almost all who have distinguished themselves in Otology during the last 50 years were his disciples. His artistic talents were used to great advantage in teaching; it was his habit to draw a sketch of each case that was demonstrated, so that the student could recognize and properly indentify the lesions.

Politzer's great work as an investigator and a pioneer, as a clinician and an operator, as a writer and a teacher was fittingly crowned by his great two-volume History of Otology, published in 1907 and 1913.

It is not possible for one to indicate the peculiar importance of Politzer's work more clearly than to point out the position which the Vienna School of Otology has held and still holds, chiefly because of the methods and work, the teaching and the influence of Adam Politzer. It is to this man to whom every one of us is so deeply indebted, that we feel it proper to pay a tribute of gratitude and respect.

MEETING OF WESTERN SECTION, HELD IN SAN FRANCISCO, CALIFORNIA, FEBRUARY 23rd AND 24th, 1922, UNDER THE CHAIRMANSHIP OF DR. GRANT SELFRIDGE, OF SAN FRANCISCO, CAL.

THE TONUS EFFECTS OF THE CRISTAE AND OF THE MACULAE.*

By S. S. MAXWELL, Department of Physiology of the University of California.

As the result of numerous investigations on the physiology of the labyrinth, Ewald arrived at his celebrated tonus theory. He laid emphasis upon the discrimination of two parts, the auditory labyrinth and the tonus labyrinth, with distinct and very different functions. By many beautiful experiments he established beyond question the fact of an influence of the labyrinth upon the tonus of many of the body muscles. His theoretical deductions were less happy. He assumed the presence of continuous ciliary activity on the part of the hair-cells and this ciliary movement was in some way associated with muscle tonus. His attention was almost wholly centered on the canals and ampullae; the otoliths were dismissed with some *a priori* considerations.

The equilibrium reactions were subordinate to the tonus effects. The ciliary activity of the hair-cells was augmentd by an increased flow of endolymph in one direction with a consequent increase of their tonic influence and hence a contraction of certain muscle groups, while an opposite flow of the current caused a checking of the movement of the hair-cells and an inhibition of tonus. We do not yet know what tonus really is nor how it is excited through the labyrinth. It is better to be content with the facts than to assume an unnecessary hypothesis.

Ewald's original conception of the tonus labyrinth applied only to the canals and ampullae. Later his pupil Ach (Ueber die Otolithenfunktion und den Labyrinthtonus. Arch. ges. Physiol., 1901, lxxxvi, 122.) spoke also of an otolith tonus, but for its existence he gave no clear evidence. Through a series of painstaking investigations carried on for more than a decade Magnus and de Kleijn have drawn attention to a most instructive

*Read by Invitation.

array of details concerning the tonus of the various muscle groups and the dependence of their tonus on various factors, chief among which is the influence of the labyrinth. They have shown, for example, that in the rabbit, when the disturbing and inhibitory effects of the cerebral hemispheres are excluded, definite tonus changes of the muscles of neck, trunk, limbs, and eyes take place with each change of position of the body. These tonus changes can be excited through different stimuli which normally coöperate and re-enforce each other. Thus when the rabbit is placed upon its side, the labyrinth gives rise to excitations which cause an unsymmetrical distribution of tonus in the neck muscles so that the head is brought into a position of symmetry with reference to the lines of gravitational force. The altered position of the head is now asymmetrical with reference to the trunk; and the changed pressures and tensions in the joints or muscles of the neck act reflexly to bring about tonus changes in the muscles of the trunk and of the limbs in such way as to raise the body into the normal posture. In addition to these sources of afferent impulses contact stimuli arising from the side on which the body lies, tend also to cause the lifting of the head and, indirectly, the raising of the body into a position of symmetry.

De Kleijn and Magnus have, on the basis of their observations, drawn a sharp distinction between reflexes which they consider to be excited by movement and those which are excited by position. This is essentially the old distinction of dynamic and static reactions. These authors have assumed that the dynamic reactions, excited by angular accelerations, have their origin in the cristae of the canals, and that the posture reflexes, on the other hand, depend upon the state of excitation of the maculae. According to this conception the tonus effects, assigned by Ewald to the ampullae, are chiefly dependent on the maculae with their otoliths.

There is no *a priori* reason for assuming the existence of static functions apart from dynamic. The excitation which brings about a posture reflex must be dynamic as well as static; the muscles must cause a movement in order that there shall be a new position to sustain, and hence the difference between dynamic and static is only a difference of the presence or absence of a mechanism for the continuance of the excitation and so of the state of muscular tonus. If this conception is correct we

should expect to find that both cristae and maculae are capable of maintaining tonus. In case there is a real physiological difference in the kind of excitation aroused in the cristae and in the maculae, then, according to the conception of the latter as the source of posture reflexes, we should expect to find tonus effects only from the maculae. The certainty with which either ampullae or otoliths can be removed in the dogfish makes this animal especially useful for the investigation of the question.

When one labyrinth of the dogfish is completely destroyed, a difference of tonus on the two sides of the body is evidenced by the unsymmetrical position of the body, eyes, and fins. Loeb (Ueber Geotropismus bei Tieren. Arch ges. Physiol., 1891, xlix, 175.) saw that when the otoliths were removed from one ear the position resembled that of an animal with the 8th nerve cut. Lee (A study of the sense of equilibrium in fishes. J. Physiol., 1894, xv, 311.) stated that when the ampullar branches of the 8th nerve were sectioned on one side, an unsymmetrical position resulted; the body was inclined to the operated side, the eye on the operated side was depressed, and the eye on the other side elevated. He also saw in animals in which he had washed out the otoliths from one ear a similar asymmetrical position. If we may judge from these observations, tonus effects which are not merely momentary but sustained, can originate in both the cristae and the maculae. I have investigated this matter farther, using the more exact extirpation methods which I described three years ago. (Labyrinth and Equilibrium, J. Gen. Physiol., 1919, ii, 123.) During the past summer the following four classes of experiments were carried out.

(1) When all three ampullae are removed from the right ear of a dogfish, the asymmetrical positions of body, eyes, and fins are very similar to those assumed when the whole labyrinth has been destroyed; namely, the body is inclined to the right, the right eye is depressed and the left elevated, the dorsal fins are turned to the left, and the paired fins on the right are elevated while those on the left are depressed. In this condition, the animal's activities seem little affected; it goes deeper or to the surface, turns to the right or to the left like a normal specimen, although the asymmetry is permanent. If now we remove the three ampullae from the left ear, the asymmetry disappears, and the condition is established which we have previously described. The animal maintains its equilibrium,

rights itself when turned over in the water, and shows normal, but somewhat weakened compensatory movements to rotations in all planes except the horizontal. These reactions show that the otolith-organs were not injured by the operation. So long as the ampullae were present in one ear and missing from the other, the asymmetrical placings of the eyes and fins gave evidence of a continuous tonic influence from the ampullae of the sound ear.

(2) All the ampullae were removed from both ears of the dogfish and after making sure that no asymmetrical injury had been done to the labyrinths, as shown by the normal posture of the animal, the otoliths were removed from the right ear. The animal continued to maintain fairly well its equilibrium in water, righted itself when turned over, and showed hardly any noticeable asymmetry of position. This makes it probable, in fact almost certain, that the asymmetry seen by previous observers on washing out the otoliths from one ear was, in reality, due to the incidental injury to or destruction of the connections of the ampullae with the vestibular structures, and was due mainly or wholly to a lack of balance of ampullar, not of otolith, tonus effects.

(3) We remove the ampullae from the right ear and observe the degree of asymmetry which follows the operation; if we now complete the destruction of the right labyrinth by washing out the otoliths, the asymmetry is not noticeably, or but very slightly, increased. Unfortunately no reliable method of determining quantitatively the degree of asymmetry could be hit upon.

(4) When the otoliths are removed from one ear with a minimum of injury to other structures the results are as described in (2) above. An asymmetrical position rarely occurs; but when it does it is probably in a specimen whose utriculus and sacculus have been injured in the operation.

A comparison of the four classes of experiments just described makes it evident that the ampullae are the seat of continuous tonic influences. When the ampullae of one ear are lacking, a tonus difference appears on both sides of the body. It would be incorrect to assume, as has been done by many writers, that the cristae of each labyrinth have a predominating influence on one side of the body. The asymmetry which occurs after one sided extirpation is due to an influence on muscles on both sides of the body in such way that the tonus of muscles of one

side is decreased while that of the antagonists of the corresponding muscles on the other side of the body is also decreased. When, for example, the ampullae are removed from the right ear, the muscles which lower the right pectoral fin and the muscles which raise the left pectoral act more weakly than normal with the result that the fin on the right side is elevated and its fellow on the left is depressed.

The question of the tonic effect of the otoliths is not so easy. At first sight it might seem that the almost complete absence of asymmetry after destruction of the otoliths in one ear shows a lack of tonic influence of the maculae. It must be remembered, however, that displacements of the otolith of the one ear can cause compensatory movements in both directions; and that these movements, in the rays, and apparently also in the dogfish, are brought about alike by displacement of the otolith in either the right or left ear. (Labyrinth and Equilibrium. III. The mechanism of the static functions of the labyrinth. J. Gen. Physiol., 1920, iii, 157.) From this it would appear that asymmetrical tonus effects may be produced by the macula of the one ear. In this case an asymmetrical operation would not give rise to differences of tonus in the homologous muscle groups on the two sides of the body, and hence we could not by this method arrive at conclusions in regard to otolith tonus in general.

The above described experiments then give us positive evidence of the tonic influence of the ampullae but are not capable of solving the problem of otolith tonus. De Kleijn and Magnus (Ueber die Funktion der Otolithen. I. Mitt. Otolithen bei den tonischen Labyrinthreflexen. Arch. ges. Physiol. 1921, clxxxvi, 61.) have attempted to find the solution in an interesting manner. As a basis for their work a careful determination was made by de Burlet and Koster (Zur Bestimmung des Stands der Bogengänge und der Maculae acusticae in Kaninchenschädel. Arch. f. Anat., 1916, 59.) of the form and orientation of the otoliths of the ear of the rabbit. From the data thus obtained a model was constructed which could be used to show the position of each otolith and macula, or otolith membrane, for every position of the head. Assuming that the labyrinth posture reflexes originated in the maculae, they studied the tonus of various muscle groups for the different positions of the head and sought to correlate these with the orientation of the otoliths in each position. The following will serve as an illustration of the method:

In a decerebrated rabbit the extensor muscles of the limbs are in an especial state of tonus, "decerebrate rigidity." This tonus depends upon, or is strongly influenced by, the labyrinth. It is found by rotating the body around the transverse axis that there is one and only one position of the animal at which the tonus of the limbs is at a maximal, namely, when the animal is in the position back downward with the line of the mouth-opening horizontal or elevated not more than 45 degrees. There is also another position in which the tonus is at a minimum, and this always differs from that of maximum tonus by 180 degrees. Comparing these positions with the planes of the different otoliths no definite relations are found for those of the sacculus. The otoliths of the utriculi, on the other hand, lie nearly in one plane, and are so situated that when the extensor tonus is at its minimum the otoliths are horizontal and resting upon the maculae, when the tonus is at its maximum the otoliths are horizontal but hanging from the maculae. Assuming that the tonus changes are actually excited from the maculae, de Kleijn and Magnus conclude that it is not the *pressure* of the otolith which acts as the stimulus, but on the contrary the *pull* or *tension* which it exerts. This conclusion reached by an indirect method, is in close accord with what has been proved by the writer through direct experiment upon the labyrinth of fishes.

Applying a similar method of analysis through the study of maximal and minimal tonus for various muscle groups, eyes, limbs, neck, trunk, etc., these authors have assigned specific tonus functions to the otolith of the utriculus, to the larger portion of the saccular otolith, and to a smaller, angular portion of the same otolith, lying in a different plane. For all the positions of all the otoliths the conclusion was reached that the maximal excitation occurs when the *macula is horizontal, and the otolith hangs below it.*

When we compare the conclusions arrived at by de Kleijn and Magnus on the functions of the different otoliths with the results obtained by the writer, a marked contrast appears in the role of the otoliths of sacculus and utriculus in the rabbit and in the fish; for in the fish all the position reflexes can be obtained from the otolith of the utriculus alone. The same may be true in the rabbit since the method of study through maximal and minimal tonus depends upon *a priori* considerations, and *a priori*

reasoning has led to a multitude of misconceptions before in the study of labyrinth function. De Kleijn and Magnus have wisely remarked that their conclusions can be applied at present only to the rabbit. The relations in other mammals remain to be worked out; while in animals with three otoliths in each ear, the individual functions must be different. We may agree with the last statement only we should say "may be", not "must be" different.

CERTAIN GENERAL CONSIDERATIONS UNDERLYING THE CARE OF ACUTE MASTOIDITIS.

By EUGENE RICHARDS LEWIS, M. D., Los Angeles, Cal.

What I have to present for your consideration is the outcome of many years personal experience with what might be called 'para-surgical' measures for dealing with acute surgical conditions. My object in presenting this, is to set forth certain personal convictions of mine, and to bring out the views of others, in the hope that we may profit mutually. I have chosen to discuss these measures in connection with acute mastoiditis, for a distinctly personal reason, and because I believe the practical value of these measures is particularly apparent in this condition.

As specialists in otology, it behooves us to study the common failings of specialists if we aspire to higher attainments. The most common failing of specialists seems to me to be narrowness—not the narrowness of ignorance, or lack of interest, but that naturally resulting from the higher evolution specialism in all fields. This tends toward intense concentration of the individual upon his own particular field, coupled with more or less meticulous observance of limits not to be transgressed out of respect to the work of others bordering on, but recognized as separate from, his own.

The best interest of our patient must always be paramount, and under certain conditions it may become necessary to disregard other considerations, and to trespass beyond the bounds of otology. The Physician in us on these occasions, must be given precedence over the Specialist.

When we undertake the care of a case of mastoiditis, we assume certain definite responsibilities; we owe to our patients more than a familiarity with the anatomy, physiology, special pathology and operative technique of otology. We owe more than a general understanding of the bearing of respiratory, circulatory, renal, digestive and nervous system conditions. There exists a larger, more fundamental mass of biologic knowledge which is of the greatest value to our patients. I believe it incumbent upon us to utilize this if we are to discharge our obligations to the full.

In no class of cases is this of more practical applicability than in acute mastoiditis. In many of these cases I believe it possible to conduct the case with safety to the patient in such a manner as to attain a successful result without operation. I speak with such fervor in favor of non-operative relief of this condition, for the reason that I have experienced operative cure and sequelae and have learned first hand, the not generally appreciated details of post-operative living with a healed mastoid. I do not propose to inflict you with a recital of too many details, but let me assure you that it is a condition devoutly to be undesired. Aside from one or two yearly frank infections of the tympano-mastoid cavities, varying from mild attacks covering a few days to severe ones of ten to twenty days, innumerable petty, but annoying, conditions have taken up their life with me since my operation. Among these I might mention easily upset intra-tympanic pressure, low-grade irritative or inflammatory spots in or about the incision, apoplectiform tinnitus attacks which are often stopped by jarring intra-tympanic relations through swallowing, inflation or similiar procedure. It reminds me often of the way a faulty light goes on and off when the fixture is jarred. I mention a few of these annoyances just to give you some idea of their character. This is true of what always had been my better ear during a lifelong series of recurring acute ear troubles.

I have observed in many other otologists the same attitude toward this condition that I possessed before I acquired my intimate acquaintence with it. Without special thought, I had taken it for granted as a closed incident when my mastoid cases had healed after successful operation. I have found, on the contrary, that operation is like a wedding—it is not the culmination of events, but the beginning.

Six years after my own operation, I published a study of this subject entitled "Cellular changes during and after Acute Mastoiditis" in which I incorporated some of my thoughts and experiences. This paper contains some further thoughts and convictions, which have crystallized during the decade following my former article. Details of diagnostic or operative methods form no part of this discussion.

During the past sixteen years, I have become more and more impressed with the necessity of devoting special attention to the condition of the patient—entirely aside from the upper

respiratory region and mastoid. I do not refer to the routine, examination of the urine, heart, lungs, circulatory and nervous systems; these naturally take their place with the X-Ray findings and examinations of nose, throat, cervical region, teeth and blood as usual collateral observations. I refer rather to study of the kind and the condition of the individual—robust or delicate, brave or cowardly, strong or weak, excitable or apathetic, good risk or poor risk. This resolves itself practically into an attempt to study the kind and condition of tissues with which we must deal.

The patient as an individual is only an aggregation of cells. Whereas our understanding of bio-physics and bio-chemistry is far from ample, we should therefore all the more make use of whatever *is* known if we are to do full justice to the patient.

The essentials of living cells include: nucleus and its limiting membrane, protoplasm of cell body, with its limiting membrane. Granted that these are maintained under proper environmental conditions, life is assured.

The studies of Loeb, Mott and many other physiologists have proven that cells—not unicellular organisms but cells originally occurring in highly organized animals, can be kept alive for years by placing them under artificial environment. These conditions must supply four essentials namely: water, alkali, heat and oxygen. The water is accessory as a solvent and medium of interchange between cells and environment; the limiting membranes insulate nucleus and protoplasm and allow endosmosis and exomosis; probably also rise and fall of potential (or cellular energy). Alkali is required for the protoplasmic potential in the chemistry of the cell, counterbalancing the acid potential of the nucleus. Heat is necessary to conserve specific cellular energy generated by the cell processes. Oxygen is particularly needed to maintain the acid potential of the nucleus.

Let us consider what is happening to the cell during acute infection; rapid loss of water interferes with normal delivery to the cell of requisite salts and oxygen, and with normal removal from the cell of waste material including carbon dioxide; lacking normal supply of salts, the alkali content of the cell body falls; sub-oxidation reduces the acid potential of the nucleus; production of energy diminishes; rapid dissemination of heat still further dissipates energy, and fatigue, exhaustion or death of the cell ensues.

Studies of infection have established certain additional chemical and physical facts which we must keep in mind constantly if we are to bring to bear everything possible in fighting disease. As a general principle in infectious processes, it seems that local or general hyper-pyrexia must be regarded as mass cellular reaction to chemical influence, producing,—(1) Alteration of the peri-cellular fluid environment or (2) alteration of the intra-cellular potential, as a consequence of precipate ion exchange between positive and negative potentials of nucleus and cell body. The first condition is represented by the dessication encountered early in acute infections, characterized by nausea, vomiting, dry tongue, intestinal disturbance, highly concentrated urine, dry skin, and mucous membranes. The second condition is represented by the irritation stage, increased activity of reflexes, hyperesthesia, insomnia, flushing, mental exaltation or delirium, pre-comatose state of diabetes,—the general irritation symptoms seen early in acute infections. Either of these two conditions,—rapid abstraction of water or profound alteration of intra-cellular potential,— or the combination of the two, is naturally followed by a state of impaired cell activity amounting to fatigue, exhaustion or death, depending on the degree.

Infectious processes are not the only agencies which may occasion such cellular states; exercise, starvation (especially fluid starvation) lack of rest, emotions, certain poisons such as strychnia and alcohol, bring about similiar states. Infections differ from all of these in that they tend to selfperpetuation of the underlying cause; the tissues are invaded by *living* poison-producers, which maintain a constantly renewing supply of all poison. The inflammation which results represents the battle between *living* things—cells and bacteria. Cessation of an infectious process occurs only when the invaded cells have conquered and put to death the invading bacteria. No such victory can be won without deaths on both sides. Under natural conditions, after recovery from infection, the field is littered with dead—bacteria and cells. The bacteria and cells which have succumbed may be carried off by the circulation and the field cleared in that way, or they may constitute such a mass that the circulation is unable to dispose of it. Sloughing or encysting then occurs and the mass is either thrown off or coffer-dammed off from the living cells surounding it. Surgical intervention at the proper time relieves the situation with great

advantage to the local and general condition. It seems safe to say that surgery never brought an infection to its end,—only cell activity can accomplish this. Surgical procedures must be looked upon as adjuncts to, but always subordinate to, the main mechanism of protection and survival, which is cell activity.

Considering this brief epitome of cellular changes under normal and abnormal conditions, let us turn our attention to the aggregation of cells, the patient himself, to see what we can do to help him during an acute infection, such as acute mastoiditis. Even if we are unable by any general measures to avoid operation in certain cases, can we not reduce the extent of necessary surgery, and set the scenery for good operative recovery? In other cases, can we not avoid operation entirely?

I do not propose to attempt to formulate concrete suggestions for applying the foregoing to acute mastoiditis. Each case requires individual consideraton. In general however, certain routine measures seem safe, logical and valuable. It would seem that the first thing to do in trying to be of help in this situation is to supply fluid, alkali, heat and oxygen to the cells.

During the past ten years, I have come to look upon fluid as the most valuable of therapeutic adjuncts in the conduct of these cases, regardless of surgical details. My rule of thumb is "An ounce per day per pound of body weight" by mouth or hypodermoclysis.

Heat, if actually applicable to the tissues affected, offers a definite physical entity of advantage to the patient, in that it introduces from without, a quantum of energy at the place where normal generation of energy is impaired. The vitality of the affected cells is the crux of the pathologic situation, and life or death of certain essential cells may depend upon apparently minor differences in local energy conservation. I regard it as a physical mistake to abstract heat under such circumstances.

The use of salts, such as citrates or acetates and bicarbonates in doses approximating a grain per day per pound of body weight has been my practice. Blood transfusion offers a measure of especially great value in severe emergencies.

Should operation prove necessary, we should use the minimum of anesthetic, avoid removal, destruction or traumatism of tissues beyond the minimum required to establish adequate drainage. After establishing adequate drainage, it is safe to be conservative of questionable tissue, leaving it to determine its own viability.

MEETING OF THE MID-WESTERN SECTION, HELD IN DENVER, COLO., FEBRUARY 14, 1922, UNDER THE CHAIRMANSHIP OF DR. JOHN M. FOSTER, OF DENVER, COLO.

DIFFICULTIES IN INTERPRETATION OF TESTS OF THE VESTIBULAR MECHANISM.

By FRANK L. DENNIS, M. D., Colorado Springs, Colo.

There are some points in the interpretation of the results of examination of the vestibular mechanism which are still perplexing, notwithstanding the large amount of work which has been done along this line all over the world.

I refer particularly to the significance, in some cases, of failure of the vertical canals to respond after stimulation, the anomalies of past-pointing and to the instances which arise, in which it is sometimes difficult or impossible, owing to the contradictory nature of the findings, to come to a definite conclusion as to the diagnosis. Indeed, we are, at times, puzzled to know even, whether we are dealing with a central or a peripheral lesion.

Some cases present a clearcut picture and we can easily make a diagnosis. For instance, the angle lesion syndrome is pathognomonic; lack of vertigo responses after stimulation points to posterior fossa; spontaneous vertical nystagmus means central lesion; perversion of nystagmus, especially after douching, points to brain stem lesion, either direct or from pressure; conjugate deviation after stimulation speaks for supratentorial affection. It is those cases which show aberrations from classical pictures which give us trouble and make it difficult to fit the parts together into a diagnostic whole.

The vestibular tests are a tremendous contribution to our diagnostic armamentarium and, as they are used more and more, and, as more accurate knowledge is acquired from studies of cases, they will become of still greater usefulness. What is needed now is more carefully studied and reported cases and especially studies of postmortems in connection with vestibular findings. The cases reported from the Mayo Clinic

by Lyons, are excellent examples of what I mean by careful reporting of cases. If I might criticize that report, I should refer to the fact that the hearing tests are not clear and one cannot always follow his reasoning in his analysis of the findings. I should urge everyone doing this work to report his cases, even though the clinical diagnosis cannot be substantiated by surgical or necropsic findings.

Uniformity of technique and careful attention to details in making the tests are highly desirable. We do not know what, if any, difference, departures from the standard technique makes. Therefore, I believe we should adhere rigidly to the 10 turns in 20 seconds for the production of nystagmus; 10 turns in 10 seconds for the estimation of vertigo and the measurement of past-pointing; water at exactly 68° for the douching and if any other temperature be used, state what it is; and especially should careful attention be paid to the position of the head of the patient. If this point is neglected, it is quite possible to get apparent perversion or inversion of the nystagmus after stimulation.

Of no less importance is a detailed history of the case particularly as regards the symptoms referable to the vestibular mechanism, such as vertigo, tinnitus, deafness, staggering, falling, nausea and vomiting, and *their occurrence in relation to each other*. This latter should be emphasized.

We want the vestibular tests to give us answers to the following questions:

1. Is the lesion toxic (functional or organic?)
2. Is it organic?
3. Is it central or peripheral?
4. If central, where is it?

I think they will enable us to answer these questions in a large proportion of the cases, especially if the findings are correlated, as they should be, with the results of other examinations, such as neurologic, ophthalmologic, etc.

Intracranial localization is admittedly one of the most difficult things in medicine. It is a great contribution to our diagnosis if, in any case, we can say: "This lesion is central; it is infra- or supra-tentorial and on the right or left side," as the case may be. At times, these tests will afford even more definite and minute localization data but we do not expect too much of them yet nor are they capable of diagnosing the nature of the lesion, except in some cases of cerebrospinal syphilis.

Fig. 1. Photograph of Brain, Case 1.

We so often find in various types of cases, that the responses from the vertical canals are absent after douching. It is well known that the verticals are much less resistant to deleterious influences than the horizontals, and are the first to fail; but, nevertheless, loss of their responses in the presence of responses from the horizontals, needs careful scrutiny in order to estimate its significance and I, personally, am often puzzled to properly evaluate this finding. According to the theory of neuraxial differentiation of the pathways from the horizontal and the vertical canals as brought out by Jones and Fisher, it is quite true that it is possible to have a lesion that will block impulses traveling along the vertical pathways and leave the horizontal pathways intact. But, we must be sure, first, that we have an intracranial condition present. Not all cases which show this phenomenon are instances of central lesion located in a spot in the brain where this blocking could occur. Other factors must be taken into consideration, viz: First, the responses from the horizontals must be good; otherwise we may be dealing with something which affects all of the mechanism, (a peripheral lesion for example) but with the effect most marked on the weakest part, i. e., the vertical pathways.

Second, a distant intracranial lesion can produce it by exerting pressure. In this instance the verticals of both sides are apt to be affected.

Very probably when a new edition of Jones & Fisher's book appears, this point will be discussed and case histories will be cited that will shed much light on this phenomenon.

Past-pointing is another sign which we need to study carefully in order to ascertain its meaning. It has frequently been my experience to fail to get the proper past-pointing responses after stimulation in cases where other signs do not point to central lesion. I consider it the most unreliable of all the responses. I apprehend that the reason is that pointing, being a voluntary act and under the guidance of the will of the patient, is often thereby modified. We know that past-pointing is one of the manifestations of vertigo and we know, further, that an individual can, by practice, control the manifestations of vertigo, as was so completely demonstrated among aviators during the war.

At the same time, let us not forget that past-pointing, both spontaneous and after stimulation, may be pathologic and of great significance, especially when corroborated by other signs.

Chart I

Mrs. R
Dec. 23, 1920

TESTS OF THE VESTIBULAR APPARATUS

NYSTAGMUS	SPONTANEOUS	POINTING RIGHT	POINTING LEFT
Looking to RIGHT ←	Shoulder from above	8" to L	4" to R
Looking to LEFT →			
Looking UP ↑ ↶	Nystagmus PRESENT Vertigo PRESENT Past-pointing INWARD		
Looking DOWN ←	Falling TO LEFT AND BACKWARD Romberg SWAYS TO LEFT AND BACKWARD		
LOOKING STRAIGHT AHEAD ←	Turning head to right SAME Turning head to left SAME Attempt to overthrow		
	TURNING		
To RIGHT → Amp. GOOD Duration 31 Sec.	To RIGHT Shoulder from above	20" to R 2 R	10 to R 2 R
	Nystagmus SLIGHTLY PROLONGED Vertigo 20" Past-pointing CORRECT	2 R T	T
To LEFT ← Amp. GOOD Duration 28 Sec.	To LEFT Shoulder from above	10" to L 10" L	T to T
	Nystagmus NORMAL Vertigo 21" Past-pointing NONE WITH LEFT HAND	4" L	T
	CALORIC		
Douche RIGHT → Amp. GOOD After 1 min. 8 sec.	Douche RIGHT Shoulder from above	T to	to R
	Nystagmus PERVERTED Vertigo NONE Past-pointing NONE WITH R. H. Falling		
Head Back → Amp. GOOD	FALLS TO RIGHT	20" to R	to R
Douche LEFT ← Amp. SMALL After 1 min. 12 sec.	Douche LEFT Shoulder from above	to L	to L
	Nystagmus PERVERTED Vertigo NONE Past-pointing CORRECT Falling		
Head Back ↶ ↴ ← Amp. FAIR		to L	T to

From Dr I. H Jones' Equilibrium and Vertigo.

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Mrs. R
12-27-20

Char. II

TESTS OF THE VESTIBULAR APPARATUS

NYSTAGMUS	SPONTANEOUS	POINTING RIGHT	POINTING LEFT
Looking to RIGHT	Shoulder from above		
Looking to LEFT			
Looking UP	Nystagmus Vertigo Past-pointing		
Looking DOWN	Falling Romberg Turning head to right Turning head to left Attempt to overthrow		

	TURNING		
Head Back To RIGHT 0 Amp. Duration Sec.	To RIGHT Shoulder from above	to	to
	Nystagmus Vertigo Momentary - slight nausea Past-pointing Falls to left without raising head		
To LEFT 0 Amp. Duration Sec.	To LEFT Shoulder from above	to	to
	Nystagmus Vertigo Past-pointing Falls to right without raising head		

	CALORIC		
Douche RIGHT ↗ Amp. good After min. 50 sec.	Douche RIGHT Shoulder from above	to R	to L
	Nystagmus Reverted Vertigo Past-pointing Opposite spontaneous Falling		
Head Back → Amp. good		to R	to L
Douche LEFT ↖ Amp. Fair After 1 min. 7 sec.	Douche LEFT Shoulder from above	to L	to L
	Nystagmus Reverted Vertigo Past-pointing Falling		
Head Back ← Amp. Small		to L	to L

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Mrs. R
1-23-21

Chart III

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT	Shoulder from above		
Looking to LEFT	Nystagmus		
Looking UP	Vertigo		
	Past-pointing		
Looking DOWN	Falling		
	Romberg		
	Turning head to right		
	Turning head to left		
	Attempt to overthrow		

TURNING

To RIGHT	To RIGHT		
Amp.	Shoulder from above	to	to
Duration Sec.			
	Nystagmus		
	Vertigo		
	Past-pointing		
To LEFT	To LEFT		
Amp.	Shoulder from above	to	to
Duration Sec.			
	Nystagmus		
	Vertigo		
	Past-pointing		

CALORIC

Douche RIGHT ↗	Douche RIGHT		
Amp. Good	Shoulder from above	to	to
After min 40 sec.	Nystagmus Perverted		
	Vertigo		
	Past-pointing		
	Falling		
Head Back →		to	to
Amp. good			
Douche LEFT ↙ ↶	Douche LEFT		
Amp. fair	Shoulder from above	to	to
After 1 min 20 sec.	Nystagmus Mixed + Perverted		
	Vertigo		
	Past-pointing		
	Falling		
Head Back ↶		to	to
Amp. Small			

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At least, the above criticisms are an honest expression of my convictions after conscientious study of a number of cases whom I have submitted to the turning and douching tests.

I wish to report a few cases illustrating some of these points. First, there are two of proven central organic lesion in which the vestibular tests correctly localized the trouble. Then, follow four cases in tuberculous subjects, in two of whom the ears were tuberculous and in the other two of whom there were findings, difficult of interpretation. The question arises, "Is there a toxemia from tuberculosis which is capable of affecting the vestibular apparatus?" Further study may give an answer to this question. The last case is one in which brain abscess was suspected but a radical mastoid operation partially cleared up the symptoms and signs. The findings from subsequent examinations show a partial return of responses which were lost.

Case Reports.

Case 1. Mrs. R., age 62. Both breasts removed seven years ago for carcinoma. No local recurrence. Dizzy for four months, coming on gradually. Totally deaf in right ear for years; hard of hearing in left ear; staggers and falls at times; head noises; no headache. Examined Dec. 23d, 1920:

Ears: Rt. M. T. normal except for slight retraction. Lt. M. T. Dull, retracted, no light reflex.

Hearing: Right ear deaf for all forks, Galton whistle and voice. Left ear. Hears C. V. fairly well, C64 O. Galton 1.5. Weber to left (the hearing ear).

Tongue: Deviates slightly to the left. Right corner of mouth droops; paresis of right arm; walks with wide base.

Vestibular Examination:

Chart 1. Spontaneous: Combined rotary, vertical and horizontal nystagmus in all positions of the bulb. (Note horizontal nystagmus to right on looking straight ahead); inward past-pointing, both arms; Romberg; falls to left and backward with any position of head.

On turning had good reactions from horizontals in nystagmus, vertigo and past-pointing except touched with L. H. (Note spontaneous).

On douching the right ear, vertical canals, there was a good but perverted nystagmus, slightly delayed, no vertigo and the spontaneous past-pointing to the left with R. H., was overcome. Horizontal canal: good and correct nystagmus and past-pointing.

On douching left ear, again we see perverted and poor nystagmus from vertical canals, no vertigo and past-pointing to left with both hands. Here the spontaneous past-pointing with the L. H. was distinctly overcome. Horizontal canal: mixed rotary and horizontal nystagmus.

The striking points are the spontaneous nystagmus, especially the vertical; past-pointing, positive Romberg and perverted nystagmus after stimulation, all indicating first, central lesion and next pressure on or lesion of the brain stem.

C.M
7-1-21

Chart IV

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS

Looking to RIGHT ←

Looking to LEFT ←

Looking UP O

Looking DOWN O

Shoulder from above

Nystagmus To right
Vertigo
Past-pointing
Falling
Romberg
Turning head to right
Turning head to left
Attempt to overthrow

POINTING

RIGHT	LEFT
4" to R	T
unable to control R.H. On upward motion, deviates to left.	

TURNING

To RIGHT
Amp.
Duration Sec.

To RIGHT
Shoulder from above — to — to

Nystagmus
Vertigo
Past-pointing

To LEFT
Amp.
Duration Sec.

To LEFT
Shoulder from above — to — to

Nystagmus
Vertigo
Past-pointing

CALORIC

With head back

Douche RIGHT →
Amp. Wide - slow
After min. 60 sec.

Douche RIGHT
Shoulder from above — to — to

Nystagmus
Vertigo
Past-pointing
Falling

Head ~~Back~~ Upright ↷
Amp. poor

very poor reaction — to — to

Douche LEFT ←
Amp. good
After min. 30 sec.

Douche LEFT
Shoulder from above — to L — to T

Nystagmus
Vertigo O — no nausea
Past-pointing
Falling

Head Back ←
Amp. Wide, quick

to L — to

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Mr. M
July 2nd 1921

TESTS OF THE VESTIBULAR APPARATUS
Chart V

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT	Shoulder from above		
Looking to LEFT			
Looking UP	Nystagmus Vertigo Past-pointing		
Looking DOWN	Falling Romberg Turning head to right Turning head to left Attempt to overthrow		

TURNING

To RIGHT Amp. Duration Sec.	To RIGHT Shoulder from above Nystagmus Vertigo Past-pointing	to	to
To LEFT Amp. Duration Sec.	To LEFT Shoulder from above Nystagmus Vertigo Past-pointing	to	to

CALORIC

Douche RIGHT None Amp. After 3 min. 35 sec	Douche RIGHT Shoulder from above Nystagmus Vertigo Past-pointing	to	to Questionable
Douched with Head Back ⟶ Amp. Good After 1 min 16 sec.	Falling	to	to
Douche LEFT ↖ ↑ ← Amp. Wide After min. 46sec.	Douche LEFT Shoulder from above Nystagmus Vertigo Past-pointing Falling	to	to Questionable
Head Back ↖ ↑ ← Amp. Wide		to	to

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Mr. M.
July 6, 1921

TESTS OF THE VESTIBULAR APPARATUS
Chart VI
SPONTANEOUS
After Mastoid Operation

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT ←	Shoulder from above	3" to R	3" to R
Looking to LEFT None	Nystagmus to right; increased by sitting up		
Looking UP None	Vertigo On sitting up		
	Past-pointing To right		
Looking DOWN None	Falling to left on sitting up		
Looking straight ahead ←	Romberg — Right eye turns inward and left		
	Turning head to right — downward. Pupils react slowly to		
	Turning head to left — light. Diplopia at times.		
	Attempt to overthrow		
Cannot touch nose with right finger while eyes are shut.			

	TURNING		
To RIGHT	To RIGHT		
Amp.	Shoulder from above	to	to
Duration Sec.			
	Nystagmus		
	Vertigo		
	Past-pointing		
To LEFT	To LEFT		
Amp.	Shoulder from above	to	to
Duration Sec.			
	Nystagmus		
	Vertigo		
	Past-pointing		

	CALORIC		
Douche RIGHT	Douche RIGHT		
Amp.	Shoulder from above	to	to
After min. sec.			
	Nystagmus		
	Vertigo		
	Past-pointing		
	Falling		
Head Back		to	to
Amp.			
With head back			
Douche LEFT ←	Douche LEFT		
Amp. Wide	Shoulder from above	to	to
After min. 30 sec.			
	Nystagmus		
	Vertigo		
	Past-pointing		
	Falling		
Upright			
Head ~~Back~~ ← ↑		to	to
Amp. Good			

On 8th ← only on looking to right.

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Chart 2. Now note the changing picture: Four days later stimulation of the vertical canals of both sides showed still a perversion of the nystagmus, but it is now oblique. Note turning with head back.

Chart 3. One month later the picture changes again and we have, after stimulation, from the right verticals, oblique nystagmus to left after 40 seconds douching; right horizontal canal, normal nystagmus; left verticals, mixed oblique and rotary nystagmus after 80 seconds douching; left horizontal, rotary nystagmus (perverted). At this time the hearing in the left ear was almost gone, there was right sided hemiplegia, right facial paralysis, bilateral ptosis and external strabismus in right eye. Bilateral choked discs were reported a few days after first examination.

The vestibular examination indicates a lesion in the posterior fossa of the left side, affecting the brain stem by pressure.

Fig. I. Postmortem: A large, firm, grayish tumor was found on the left side of the pons extending forward through the forandum rotundum (which was greatly enlarged), eroding the anterior and posterior clinoid processes, and extending backward, compressing the entire brain stem and left cerebellum and making a deep depression in the under surface of the left temporo-sphenoidal lobe.

Case II. Mr. M., age 22. Was in the artillery in France and gives history of trouble with right ear ever since—a period of two years. When first seen, eight days after admission to the hospital, was very stuporous and complained of violent headache. Had complained of not feeling well for a week before entering and on the day of admission was taken rather suddenly ill with headache. Vomited three times. The neurologic examination revealed: pupils normal and react; slight edema of both discs; stiff neck; positive Kernig; positive Babinsky on left; cranial nerves negative. Temperature has varied between 99° and 100°; pulse 84.

Spinal Fluid: Pressure not increased, cell count 60, no bacteria in smear; Wassermann negative.

Blood: Leucocytes 11,600, polys 68%.

Examination, June 28th. Ears: Left M. T. normal. Right canal contains small amount of foul smelling secretion; no details of M. T. made out.

Examination, July 1st. Hearing: Hears low, middle and high C forks in both ears; whisper, one foot in right ear; Weber to left.

Chart 4. Vestibular examination: Spontaneous: Horizontal nystagmus to the right on looking to right and on looking to left. Past-pointing R. H., 4″ to right. L. H. T. Unable to control R. H. On upward motion, deviates to left.

Douching right ear: Very poor nystagmus but correct from verticals. Good reaction from the horizontal. No nausea, no vertigo.

Left ear: Perverted nystagmus from the verticals. Good reaction from the horizontal.

July 2d. Right pupil larger and fixed to light; right eye turns up, left downward.

Chart 5. Douching right ear: Vertical canals fail to respond. This change has occurred after an interval of only one day between examinations. Horizontal canal: Good reaction.

Left ear: Pathways of both horizontal and vertical canals show effects of pressure in that perversion of nystagmus is manifest. X-ray of right mastoid shows evidence of chronic mastoiditis, all cells being obliterated.

CHART VII

J. F. F.
6--16--21

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT ←	Shoulder from above	3" to R	3" to R
Looking to LEFT →			
Looking UP SUSPICIOUS	Nystagmus PRESENT Vertigo PRESENT Past-pointing PRESENT		
Looking DOWN NONE	Falling		
LOOKING STRAIGHT AHEAD O	Romberg Turning head to right NOT TESTED Turning head to left Attempt to overthrow		

TURNING

To RIGHT Amp. Duration Sec.	To RIGHT Shoulder from above	to	to
	Nystagmus Vertigo Past-pointing		
To LEFT Amp. Duration Sec.	To LEFT Shoulder from above	to	to
	Nystagmus Vertigo Past-pointing		

CALORIC

Douche RIGHT ← Amp. TREMENDOUS After 1 min. 12 sec.	Douche RIGHT Shoulder from above	6 to R	6 to R
	Nystagmus Perverted Vertigo VIOLENT Past-pointing CORRECT Falling TO RIGHT		
Head Back → Amp. FAIR		6 to R	6 to R
Douche LEFT O Amp. After 3 min. 55 sec.	Douche LEFT Shoulder from above	3 to R	3 to R
	Nystagmus NONE Vertigo NONE Past-pointing SAME AS SPONTANEOUS Falling NONE		
Head Back ← Amp. GOOD		T to	T to

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CHART VIII

J. F.F.
6--19--21

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT	Shoulder from above	T & 2 to R	T & 2 to R
Looking to LEFT			
	Nystagmus		
Looking UP	Vertigo		
	Past-pointing		
Looking DOWN	Falling		
	Romberg		
	Turning head to right		
	Turning head to left		
	Attempt to overthrow		

TURNING

To RIGHT	To RIGHT		
Amp.	Shoulder from above	to	to
Duration Sec.			
	Nystagmus		
	Vertigo		
	Past-pointing		
To LEFT	To LEFT		
Amp.	Shoulder from above	to	to
Duration Sec.			
	Nystagmus		
	Vertigo		
	Past-pointing		

CALORIC

Douche RIGHT 0	Douche RIGHT		
Amp.	Shoulder from above	T to	T to
After 4 min. 40 sec.			
	Nystagmus		
	Vertigo MOMENTARILY AS DOUCHING WAS STOPPED		
	Past-pointing NONE		
	Falling TO RIGHT		
Head Back		to	to
Amp. NONE			
Douche LEFT 0	Douche LEFT		
Amp.	Shoulder from above	T to	T to
After 5 min. 0 sec.			
	Nystagmus NONE		
	Vertigo		
	Past-pointing		
	Falling		
Head Back ←		T to	5 to L
Amp. GOOD			

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CHART IX

J. F. F.
7--6--21

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT ← (few twitches)	Shoulder from above	2" to R	4" to R
Looking to LEFT NONE			
Looking UP ↑ SUSPICIOUS	Nystagmus Vertigo Past-pointing		
Looking DOWN NONE	Falling Romberg Turning head to right Turning head to left Attempt to overthrow		

TURNING

To RIGHT Amp. Duration Sec.	To RIGHT Shoulder from above	to	to
	Nystagmus Vertigo Past-pointing		
To LEFT Amp. Duration Sec.	To LEFT Shoulder from above	to	to
	Nystagmus Vertigo Past-pointing		

CALORIC

Douche RIGHT 0 Amp. After 4 min. 45 sec.	Douche RIGHT Shoulder from above	T to	T to
	Nystagmus NONE Vertigo ON STOPPING DOUCHE Past-pointing NONE Falling		
Head Back 0 Amp.		4 to R	4 to R
Douche LEFT 0 Amp. After 5 min. sec.	Douche LEFT Shoulder from above	1/2 to L	1/2 to L
	Nystagmus NONE Vertigo NONE Past-pointing Falling		
Head Back ← Amp. GOOD	VERTIGO, SLIGHT	T to	T to

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A diagnosis of lesion in the right cerebellum, probably abscess, was made. A radical mastoid operation was done with the intention of exploring the cerebellum but, owing to an accidental wounding of the sinus wall, it was given up. The antrum and middle ear were filled with cholesteatomatous masses—no cellular structure.

The following day the general condition was much improved and the next day he was entirely rational and free from headaches, though the mental processes were somewhat sluggish. He still had spontaneous horizontal nystagmus to the right and inco-ordination of the right arm.

Chart 6. Douching the left ear with the head back, a correct and good nystagmus was produced; a mixed oblique and horizontal nystagmus to the right after putting head upright.

Five days after the mastoid operation the cerebellum was explored both in front of and behind the sinus but the abscess was not found. Following the operation, there was great improvement in the general condition; appetite returned, there was no headache, and patient sits up in bed; the temperature was normal, pulse 85 to 108, and the falling to the left had disappeared. This favorable condition persisted for six days when the headache returned, patient was drowsy, face drawn to the left, unconsciousness supervened and death occurred.

Postmortem revealed: Flattened convolutions in right hemisphere and an abscess containing half an ounce of pus in the right anterior lobe of the cerebellum. No thrombosis of the sinuses.

Comment.

It was evident that here we must have an intracranial lesion. Meningitis was at first suspected. The examination of the ears revealed a very poor nystagmus from the right verticals and a perverted nystagmus from the left verticals, while the horizontal canals reacted well. The two together indicated central lesion and the perversion pointed to pressure on the stem. On the next day this was emphasized by the total lack of responses from the right vertical canals and still more perversion of the nystagmus from the left verticals and perversion of nystagmus from the left horizontal. The condition of the right ear and mastoid, plus the history of long-standing ear trouble made abscess the most likely diagnosis; while the total ablation of the responses from the vertical canals of the affected side with evidence of pressure on the pathways of the opposite side—that is unequal effect on the verticals of the two sides—pointed to nearby lesion and hence, abscess of the right cerebellum. The spontaneous past-pointing of the right arm was corroborative. The neurologic data fitted in very well with these findings.

Case III. J. F. F., 29 years. Is quite ill with tuberculosis; has lesions in lungs, peritoneum and hip. Has had right-sided otorrhea for several months

CHART X

A. E.
6--20--21

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT 0	Shoulder from above	T	2 to R
Looking to LEFT 0	Nystagmus NONE		
Looking UP 0	Vertigo YES		
	Past-pointing WITH L. H.		
Looking DOWN 0	Falling		
	Romberg SWAYS TO LEFT		
	Turning head to right SWAYS TO BACK & LEFT		
	Turning head to left SWAYS FORWARD & LEFT		
	Attempt to overthrow		
FINGER TO NOSE, NEGATIVE.	ADIODOKOKINESIS. NEGATIVE		

TURNING

To RIGHT ⟶	To RIGHT		
Amp. GOOD	Shoulder from above	2 to R	2 to R
Duration 8 Sec.			
STOPPED SUDDENLY	Nystagmus SUBNORMAL		
	Vertigo 7 sec		
	Past-pointing CORRECT		
To LEFT ⟵	To LEFT		
Amp. GOOD	Shoulder from above	T to	T to
Duration 11 Sec.			
STOPPED SUDDENLY	Nystagmus SUBNORMAL		
	Vertigo 11 sec		
	Past-pointing NONE		

CALORIC

Douche RIGHT 0	Douche RIGHT		
Amp.	Shoulder from above	T to	T to
After 5 min. sec.			
	Nystagmus NONE		
	Vertigo NONE		
	Past-pointing NONE		
	Falling NONE		
Head Back 0		T to	T to
Amp			
Douche LEFT ↶	Douche LEFT		
Amp. SLOW--IRREGULAR	Shoulder from above	2 to L	T to
After 1 min. 50 sec.			
	Nystagmus POOR--DELAYED		
	Vertigo NONE		
	Past-pointing NONE WITH L. H.		
	Falling		
Head Back ⟵		6 to L	2 to L
Amp. QUICK--GOOD	DIZZY		

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CHART XI

H.E.D.
8-2-21.

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT 0	Shoulder from above	T	2" TO R. & T.
Looking to LEFT 0			
Looking UP 0	Nystagmus NONE Vertigo NONE Past-pointing TO R. WITH L.H.		
Looking DOWN 0	Falling Romberg 0 Turning head to right 0 Turning head to left 0 Attempt to overthrow		

TURNING

To RIGHT → Amp. VERY SMALL. Duration 10 Sec.	To RIGHT Shoulder from above Nystagmus Vertigo 0 Past-pointing	4" to R T	2" to R T
To LEFT ← Amp. QUICK, FINE. Duration 17 Sec.	To LEFT Shoulder from above Nystagmus Vertigo 0 Past-pointing	2" to L T	2" to R. 2" to R. T.

CALORIC

Douche RIGHT 0 Amp. 0 After 5 min. 14sec.	Douche RIGHT Shoulder from above Nystagmus NONE Vertigo NONE Past-pointing NONE Falling	T to	T to
Head Back → Amp. WIDE	SLIGHTLY DIZZY.	T. to	2" to L. 4" to R.
Douche LEFT 0 NONE Amp. After 5 min.15 sec.	Douche LEFT Shoulder from above Nystagmus NONE Vertigo NONE Past-pointing NONE Falling	T. to	T. to
Head Back ← Amp. IRREGULAR, WIDE	SLIGHTLY DIZZY.	T to	2" toR

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CHART XII

A. P. M.
12--16--21

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT 0	Shoulder from above	1/2 to R	T
0			
Looking to LEFT 0	12/13	T	3" to R
Looking UP 0	Nystagmus None Vertigo NOT NOW Past-pointing		
Looking DOWN 0	Falling Romberg NEGATIVE Turning head to right Turning head to left Attempt to overthrow		

TURNING

To RIGHT ⟶ Amp. GOOD Duration 17 Sec.	To RIGHT Shoulder from above	4 to R 1 R	4 to R 4 R
	Nystagmus SUBNORMAL	T	3 R T
	Vertigo 17 sec		
	Past-pointing CORRECT		
To LEFT ⟵ Amp. GOOD Duration 23 Sec.	To LEFT Shoulder from above	6 to L 2 L	6 to L 2 L
	Nystagmus NORMAL	2 R	2 R
	Vertigo 23 sec	2 L	2 R
	Past-pointing CORRECT--NOTE SPONTANEOUS		

CALORIC

Douche RIGHT 0 Amp. After 3 min. 55 sec.	Douche RIGHT Shoulder from above	T to	T to
	Nystagmus NONE Vertigo NONE Past-pointing NONE Falling		
Head Back ⟶ Amp. GOOD	DIZZY	T to	2 to R
Douche LEFT 0 Amp. After 4 min. 23 sec.	Douche LEFT Shoulder from above	2 to L	1 to R
	Nystagmus NONE Vertigo NONE Past-pointing ERRATIC Falling		
Head Back ⟵ Amp. WIDE	VERTIGO SLIGHT	2 to L	1 to R

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which is clinically tuberculous. For three weeks has had continuous vertigo (feels that he is turning to the right), with violent occipital headache, requiring morphin for relief. Frequent nausea and vomiting. Lying on the right ear or pressure above the temporo-maxillary articulation produces increase of vertigo.

Ears: Right, purulent discharge. Left, normal.

Hearing: C^{64} ½″ / 3″ — C — 4″ / 10″ — C^{4} poor / good — W. V. — 18″ — Weber to right.

Vestibular examination, June 14, 1921.

Chart 7. Spontaneous: Horizontal nystagmus, to the right and left respectively, on looking to the right and to the left with a suspicion of vertical nystagmus upward on looking up. Pastpoints to the right with both hands.

On douching the right ear (vertical canals) with water at 68° F., a tremendous perverted and inverse nystagmus with violent vertigo appeared after 72 seconds. Note that there was correct past-pointing and falling to the right. The horizontal canal showed a fair nystagmus of proper kind and direction and correct past-pointing.

On douching the left ear, there was no nystagmus from the vertical canals, no vertigo and the pastpointing was the same as the spontaneous. On putting the head back, the left horizontal gave good and correct nystagmus and both hands touched, overcoming the spontaneous pastpointing to the right.

Chart 8. Five days later the vertical canals of both sides were out of business, as well as the right horizontal, the only canal giving a reaction being the left horizontal.

Chart 9. Twelve days later, practically the same picture was presented. On the occasion of the last two examinations, a curious phenomenon was noted, viz.: after douching the right ear there was momentary vertigo at the moment of discontinuing the douching—not before.

The noteworthy observations are the perversion of the nystagmus from the right verticals with violent vertigo and yet, five days later every canal but one is unresponsive. The picture is that of angle lesion with only one factor lacking—retention of hearing in the right ear. The symptoms gradually improved and the patient left town. In a letter received a few days ago, he stated that his vertigo had disappeared but he still suffered from head aches and was doing poorly generally. In my opinion, he has a lesion in the posterior fossa on the right side near the angle. One would strongly suspect tuberculoma.

Case IV. Miss A. E. In this case, one would think the findings point to an angle lesion if only the douching tests are considered; for, we have a total lack of responses from the right labyrinth with poor responses from the left verticals and good responses from the left horizontals. However, when the history and the hearing tests are taken into account, this diagnosis is not warranted.

Hearing: Almost total deafness in the right ear. Very poor in the left ear, which is greatly improved by an artificial drum membrane.

History: Has been tubercular for a number of years. Eight years ago had a tubercular otitis in the right ear, followed by a radical mastoid operation. Healing did not take place until over two years of constant treatment had been carried out. In the meantime, tuberculosis of the left ear developed

CHART XIII

F. McK.
9--8--20

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS			POINTING RIGHT	POINTING LEFT
Looking to RIGHT	0	Shoulder from above	T	T
Looking to LEFT	0			
Looking UP	0	Nystagmus NONE Vertigo NONE Past-pointing NONE		
Looking DOWN	0	Falling NONE Romberg NEGATIVE Turning head to right Turning head to left Attempt to overthrow		

TURNING

To RIGHT ⟶ Amp. SMALL Duration 11 Sec.	To RIGHT Shoulder from above	10 to R T	6 to R 2 R T
	Nystagmus DIMINISHED Vertigo 11 sec Past-pointing CORRECT FALLING NONE		
To LEFT ⟵ Amp. SMALL Duration 8 Sec.	To LEFT Shoulder from above	12 to L T	T to T
	Nystagmus DIMINISHED Vertigo 9 sec Past-pointing FAULT WITH L. H. FALLING NONE		

CALORIC

Douche RIGHT 0 Amp. After 4 min. 15 sec.	Douche RIGHT Shoulder from above	T to	T to
	Nystagmus NONE Vertigo NONE Past-pointing NONE Falling		
Head Back 0 Amp.		2 to R	T to
Douche LEFT 0 Amp. After 4 min. 55 sec.	Douche LEFT Shoulder from above	T to	T to
	Nystagmus NONE Vertigo NONE Past-pointing NONE Falling		
Head Back 0 Amp.	NAUSEATED	T to	T to

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CHART XIV

F. McK.
9--14--21

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS		POINTING RIGHT	POINTING LEFT
Looking to RIGHT	Shoulder from above		
Looking to LEFT			
Looking UP	Nystagmus Vertigo Past-pointing		
Looking DOWN	Falling Romberg Turning head to right Turning head to left Attempt to overthrow		

TURNING

To RIGHT ⟶ Amp. GOOD Duration 9 Sec.	To RIGHT Shoulder from above	to	to
	Nystagmus DIMINISHED Vertigo Past-pointing FALLING NONE		
To LEFT ⟵ Amp. GOOD Duration 9 Sec.	To LEFT Shoulder from above	to	to
	Nystagmus DIMINISHED Vertigo Past-pointing FALLING NONE		

CALORIC

Douche RIGHT 0 Amp. After 5 min. 10 sec.	Douche RIGHT Shoulder from above	2 to R	2 to R
	Nystagmus NONE Vertigo Past-pointing Falling		
Head Back 0 Amp.		T to	T to
Douche LEFT ↶ Amp. SMALL After 1 min 35sec.	Douche LEFT Shoulder from above	2 to L	4 to L
	Nystagmus POOR & DELAYED Vertigo Past-pointing Falling		
Head Back ⟵ Amp GOOD		T to	T to

ENTIRE TEST AT ONE SITTING NO NAUSEA OR VERTIGO

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which finally became dry with a persistent perforation involving the lower and anterior half of the membrane. Her history of vertigo dates back two or three years from the time of the examination. The first very severe attack was six weeks ago, followed by another two weeks ago, in which she was nauseated and vomited. There is a constant feeling of unsteadiness and she has fits of forgetfulness in which she cannot remember, for example, where she lives but, "could go there".

Chart 10. It is to be noted that all responses from both ears are affected. This points to a bilateral peripheral lesion. What has probably happened is that a slowly progressing labyrinthitis occurred in the right ear at the time of the mastoiditis which gradually put the labyrinth out of business. This was so slow in developing that compensation took place along with this destruction so that there were no noticeable attacks of vertigo. Later, the other labyrinth is becoming involved and the vertiginous attacks are the result of irritation.

Case V. Mr. D., age 40. This is an example of total loss of responses from the vertical canals of both sides in a case of what is probably not a central lesion.

Chart 11. It is to be noted that the responses from the horizontals in nystagmus are poor, there is no vertigo and poor pastpointing.

Ears: Both membranes intact, the left retracted.

		3″	10″	fair	35′		
Hearing:	C^{64}	C	C^{4}	W. V.		Weber to right.	
		3″	10″	poor	2′		

The patient has tuberculosis, gives a history of mumps at 1 year; rheumatism fifteen years ago; vertigo two years ago, lasting two months. Wassermann was not done. The case might be a toxemia from mumps or lues. It is very difficult to estimate the significance of the loss of the vertical responses. Note the entire absence of vertigo after stimulation with a poor nystagmus from both horizontals. Also diminished hearing in the left ear and Weber to the right, or better hearing ear.

Case VI. A. P. M., age 47. This is another case with pulmonary tuberculosis with but little involvement.

He has noticed some dizziness for more than a year. Last June had quite a severe attack and had to remain in bed, but did not vomit. Had another attack two weeks ago; surrounding objects appeared to turn to the right. Has had pneumonia, mumps, "stomach trouble" and abscess of the ear several years ago.

Both drum membranes are intact.

		3″	4″	good	20′		
Hearing:	C^{64}	C	C^{4}	W. V.		Weber to left	
		3′	8″	good	30′		

Chart 12

Vestibular examination: Turning: Fairly good nystagmus, pastpointing and vertigo, although the pastpointing is erratic.

Douching (next day). No responses from the verticals. Three days later another douching test revealed practically the same result.

Comment.

It is to be noticed that the hearing is somewhat diminished in the right ear, the Weber is toward the better hearing side and there is a history of mumps. The vertical canals of both sides are out of commission with good responses from the horizontals. This is difficult to explain as there is no other evidence of central lesion. As I see it, the most plausible hypothesis is some destructive action on the fibres from the vertical canals and the cochlea of the right side by a toxic process—probably mumps. The case will be re-examined and further observed and perhaps something will be found later that will clarify the diagnosis. If some evidence of central lesion shows up, as for instance, spontaneous vertical nystagmus, we will be warranted in attributing failure of the vertical responses to that cause.

Case VII. The next case is puzzling. F. McK, age 35. May 5, 1920.

History: Discharge from right ear at intervals for twelve or fifteen years. Has had bronchiectasis for twenty years. Ten days ago, pain and discharge from right ear; tender swelling above auricle, extending almost to the orbit. Large perforation in M. T. Mucous membrane swollen and pus coming from ear. Mastoid is not tender. The signs at this time pointed to some brain complication. He vomited; mental dullness alternating with irritability; thick speech; weakness of left facial; twitching of right facial muscles; convulsion; moderate swelling of right eye grounds; rotary nystagmus on looking upward; nystagmus to the right on looking towards the left; right internus weak; past-pointing to the right with both hands; total deafness of right ear with noise apparatus in left; Weber to the left; no reaction from any canal on douching the right ear. (Unfortunately the left ear was not douched at this time.) A radical mastoid operation was done, after which all symptoms rapidly cleared up.

Four months afterwards a complete vestibular examination showed the following:

Chart 13

Spontaneous: No nystagmus, past-pointing on vertigo.

Hearing: Complete deafness in right ear.

Left ear: C^{64} 3″, C 10″. c^4 good, W. V. 15 feet.

Weber not lateralized.

The left drum membrane is intact but deformed by scars and atrophic spots.

Right ear: Healed radical operation.

Turning: Nystagmus is much diminished; pastpointing is normal except the left hand cannot be made to pastpoint to the left.

On douching there were no responses from any canals of either side except that after douching the left ear, putting the head back and then upright again, there were a few twitches of horizontal nystagmus to the left and slight nausea.

Chart 14

Sixteen months later another examination revealed: No nystagmus from the verticals or the horizontals of the right ear but in the left ear the impulses are going through. He gets a poor nystagmus from the verticals and a good response in nystagmus from the horizontal; both arms can be made to past-point in both directions.

The first examination distinctly pointed to some central disturbance. The second shows returning function on the left side and suggests that whatever was affecting these responses is ceasing to operate. A bilateral maxillary sinus operation was done during the summer preceding the second examination. A possible explanation is that the left labyrinth was being affected by toxemia from this focus and now that it is removed the responses are beginning to appear. The right labyrinth has probably been destroyed by a peripheral lesion.

DISCUSSION

Dr. Robert Levy, Denver, Colo.: Mr. President, my discussion will necessarily be very brief. I say "necessarily," because in the first place the subject is one which is as yet unsettled, and in the second place, a paper such as this, and like the one which has been read by our friend, Dr. Hemstead, cannot be discussed properly unless one sits down and digests its contents. And that leads me to say this, that in our study of internal ear cases, and in those in which we make vestibular tests, we must study very carefully our findings.

As Jones has pointed out, we should tabulate these findings as definitely and as accurately as we can, confirm them by repeated examinations, then sit down as with any problem, and study it, after which we may arrive at some reasonable conclusion. In order to do this it is necessary to have a uniform and systematic method of procedure.

It is very difficult to draw comparisons between the results that one man obtains and the results that another obtains with our present knowledge of this subject, for the reason that all men seem to be conducting their work along different lines. At any rate, they have different ways of expressing themselves; they have different charts and different methods of performing the same tests. And so if I had anything to say at all, Mr. Chairman, it would be as a plea for more uniform methods, for more uniform tabulations of results of examinations.

This very clear exposition of the subject by Dr. Dennis is gratifying. In Dr. Dennis' preliminary remarks, he very succinctly called attention to certain definite objects that we have in mind when we make a study of labyrinthine cases, and if we can bear in mind these definite points and keep them clearly before us we may be able to come to some sort of an understanding of the problem before us. As to the difficulty of diagnosis, I think there is no field that we today as Otologists are interested in that offers greater difficulties in determining the underlying lesion. There never has been anything easy in our study of these cases. Certain rules that have been laid down by the masters are rules to which there are many exceptions, and, as a matter of fact, it is the exceptions that prove the rule.

I should like to emphasize one point which has been of much help to me, and that is that in performing our tests we should use one test to check up the other; and so I have gotten the greatest benefit, the greatest satisfaction, by checking up my turning tests with my vertigo tests, and then finally checking up these two by the caloric test, either with hot or with cold water.

Dr. F. R. Spencer, Boulder, Colo. Mr. Chairman, I don't know that I have anything to add to what has been said.

I think Dr. Levy emphasized the thing which we mentioned at lunch and that is the necessity for having an opportunity to sit down and thoroughly digest these papers. Dr. Hemstead was with us at lunch, and we mentioned this in connection with his paper. By the way, we

will be glad to hear from Dr. Hemstead regarding this. I hope he will discuss Dr. Dennis' paper.

This is such a tremendous field, and so much is being added to our knowledge all the time, that is is a difficult matter to add something in particular connection with it. I think Dr. Dennis has had an unusual opportunity in his Army experience to study any number of these cases and to test a great many of them. One thing I have always felt was very important, and that is many times when we see a case which should be tested, we are busy and we do not test the case as accurately as we should, or we do not see the patient until the next day, and the result is our records are not as complete as they should be. We know that the Ophthamologist is able to give the Neurologist a great deal of valuable information by using the ophthalmoscope and in making suggestions concerning a possible diagnosis, and the same can be done with tests with vestibular apparatus, and we can add a great deal to the knowledge which we have at the present time if we will make these tests, and if we will carry out the lines of investigation which we now have before us.

Dr. Robert Levy: I would like to emphasize two points. I think I have mentioned them, but I would like to add a little to what I said. In making these tests we ought to repeat them rather frequently before we attempt to draw conclusions; and the other point is, that we ought not to make too many of the tests at the same sitting. I think we are apt to become confused, and I believe our patient is apt to give us improper responses if we attempt to complete the examination at one or two sittings. Perhaps we should first make a simple rotation test; then give the patient a rest for twenty-four hours. By going over this again we can each time add something to our original examination, and so will be more likely to get responses that are reliable than if we attempt to do it all at once. We will be less liable to become confused.

Dr. Frank L. Dennis (Closing): I just wanted to say that Dr. Levy emphasized the thing I was trying to bring out in the paper which I read, and that is uniformity of the technique. That is one of the things which is going to help us a lot in coming to our conclusions.

Both Dr. Levy and Dr. Hempstead spoke of these dicta which have been put out by Jones and Fisher not panning out. I think that is true, and I think that now if Jones and Fisher were to bring out another edition of their book they would not make such positive statements.

In regard to checking up the tests and doing them on different days, that is very important from two standpoints. One is that the patient won't stand for it in many cases, except in those cases where we do not have the production of vertigo from the tests. In some posterior fossa lesion cases the whole test may be carried out without a particle of vertigo being experienced by the patient, and under these circumstances, the tests may be carried out all at one sitting.

I am very glad indeed to hear what Dr. Hemstead has to say about these several hundred cases that he is working through. It seems to me that the difficulty which we experience now in trying to interpret the findings in these cases is because we have not enough definitely worked-out cases to study. We get many cases of angle syndromes, and cases like the two I reported here, but we do not get the great mass of cases which do not pan out. There are many things about the findings which need clearing up.

With regard to what Dr. Hemstead said about Weisenburg's theory about the differentiation of the pathways. Jones and Fisher have adopted Weisenburg's ideas in that particular; and when I was East last fall I saw a new chart which he had detailing these pathways.

DEMONSTRATION OF CASES.

By WM. C. BANE. M. D., Denver, Colo.

1. R. L. aged 25 years, the radical operation on right mastoid at 15 years of age. Occasional cleansing of cavity of crust formation. Hearing—whisper at three feet.

2. Mrs. G. A. P. aged 34 years, the radical operation on right mastoid at 18 years of age. Middle ear and antrum filled with cholesteatomatous deposits. Lateral sinus uncovered, diseased-drained and packed. On 23rd day ear quite dry, no odor, hearing then 16/60 (watch). Ear keeps quite dry. Hearing now 18/20 (whisper).

3. Mrs. J. H. aged 45 years, history of chronic disease of right antrum of Highmore. In Feb. 1921, anterior portion right inferior turbinal removed —antrum perforated—foul pus evacuated. Opening enlarged. Six days later developed acute otitis media and mastoid tenderness. Free drainage of middle ear and suction irrigation cleared otitis media and mastoiditis in a few weeks. Antrum was kept clean by irrigation. Pus disappeared. A large opening into the antrum remains.

4. Mrs. J. L. aged 47 years. Two years ago frontal headache came on without apparent cause. For last month has been almost continuous, supra orbitally on right side. Pain worse on lying down, constricting in character. Asthenopia marked. Cannot use eyes for close work. Heat locally relieves. Examination by X-ray shows sinuses clear. No pus in nasal cavity. Ewings sign present. Right middle turbinate enlarged and resting on slight ridge on external wall.

Diagnosis: Vacuum Frontal Sinusitis (right).

Treatment: Removal anterior 2/3 of right middle turbinate.

5. Mrs. E. J. Aged 49 years. Patient complained of catarrhal condition of nose with post-nasal discharge. Is extremely nervous and has dull headache, worse in forenoon and on lying down. Examination of nose showed anterior end of each middle turbinate enlarged and polypoid with purulent discharge from middle meatus. X-ray showed both antra cloudy.

Treatment: Under local anesthesia both antra opened. Four months later radical operation performed on both sides. Headaches relieved but discharge persisted. Patient is now being treated by local applications of radium with steady decrease of discharge.

6. Miss A. T. aged 20 years. Operated three weeks ago by Dr. Joseph Beck, of Chicago for radical antrum on both sides. Examination showed both antra well opened with free muco-purulent discharge. Have swabbed both sides with 1% mercurochrome and treated with radium. Discharge has ceased and except for some formation of crusts in the nasopharynx patient is apparently relieved.

GASTROSTOMY IN LARYNGEAL TUBERCULOSIS.

By CLAUDE E. COOPER, M. D., Denver, Colo.

The pitiable condition of gradual starvation, with repeated, intense pain on swallowing saliva and food; the thirst, parched mouth and throat, progressing emaciation, increasing toxemia and very evident decline, together with the oft repeated plea for help and relief; that is found in those with extensive ulcerative tubercular lesions of the larynx, has occasioned more sympathy and unfortunately less relief from the medical attendant, than most of the other serious manifestations of tuberculosis.

Our efforts to allay these distressing symptoms by relying on local anesthetics, analgesics and the internal administration of narcotics, have too often proven futile: and we have frequently watched our patient make the exit from life with great suffering and distress. Of our sympathy they have plenty; of effectual help, but very little; of cure, none. We recognize that the time has come for this one or that, when the physician must step aside unable to prevent the one thing of life which is inevitable; even though such an event is in all probability our most interesting experience. Any procedure, therefore, that might lessen the pain of these unfortunates, prolong their life free of suffering or make their exit in comparative comfort is worthy of consideration.

From a study of these cases, coming under personal observation, gastrostomy is an advance over other methods in accomplishing this end.

Untried procedures are at first quite justly applied to those cases which resist measures in more general use, and consequently only those of advanced disease,—therefore, less adapted to any remedial agent,—receive the experiment. As time passes and the cases grow in number, any method possessing merit will grow in favor and naturally be more intelligently applied.

I have five cases to report.

Case Reports.

Case 1. A woman, 33 years of age, seen with Doctor Lowen. This case was reported by him at a meeting of the City and County Medical Society, of Denver, and published in Colorado Medicine in May, 1920. She had a

well advanced bilateral lung involvement with cavitation and her larynx presented an ulcerated, hypertrophic, edematous laryngeal tuberculosis. There was extreme dyspnea, dysphagia, gradual decline, and a most distressing death not many days away.

The ordinary cocain spray and powder with cleansing solutions, sprays, etc., had been prescribed, giving the known unsatisfactory relief.

On several occasions Doctor Lowen and I considered the advisability of a gastrostomy, and on procuring the patient's consent, he performed it under local anesthesia using the Stamm-Kader method.

There was surprisingly little abdominal distress after the operation, and the shock of manipulation soon subsided. This was not great, and in importance, could not be considered a barrier to the procedure, especially in view of the relief afforded. The pain subsided, and a general comfort displaced suffering.

She showed signs of general improvement for three months, then gradually lost ground and finally died three months later, or six months after the gastrostomy, from a further extension of her throat and lung involvement.

The operation had accomplished what was desired of it. It did not cure, nor was such expected, but it did alleviate.

A physician who specializes largely in tuberculosis, after observing this case made the statement, that because of the relief obtained, he would not allow another such case to proceed without advising a gastrostomy.

Case 2. A case which I saw at the National Jewish Hospital: R. S., male, 38 years of age, Jewish.

Family history. Negative, except the death of a brother at 18 years following an operation on the larynx for relief of dysphagia, incident to laryngeal tuberculosis.

Personal history. Good health until two years ago when he contracted a cold and sore throat. The cold subsided, but his throat continued to annoy him. There was dysphagia, gradually becoming worse, cough and expectoration, which lasted for one and one half years, during which time he was afebrile. Finally he consulted a laryngologist, who diagnosed tuberculosis and sent him West.

On admission to the Hospital, he was fairly nourished; weight 133 pounds; height 5 feet 7 inches; hemoglobin 85%; white and red blood count normal; Wassermann negative; sputum positive for tubercle bacilli; urine negative.

Physical examination and X-ray showed advanced pulmonary tuberculosis of the fibroid type, with inflammatory mottling in the upper lobe of the left lung, and some mottling extending throughout the whole of the right lung.

His laryngeal condition, according to Doctor McKeon who first examined him was: "Infiltration and edema of the arytenoids, epiglottis, ventricular bands and vocal cords, with ulceration in the anterior commissure and along the left aryepiglottic fold". Speech was in a whisper. Later he had an increasing dysphagia especially for liquids and semisolids.

The usual sprays and applications—cocain, orthoform, etc.—gave no relief. Death from thirst and starvation was imminent.

I then advised gastrostomy and after several consultations ,the patient desiring it, Doctor George Packard operated under local anesthesia.

He stood the operation well; received some water through the tube eight

hours later, and the next day was given liquid nourishment.

From now on he began to improve, gained weight, and the laryngeal pain disappeared. For eight months he lived on a liquid diet, feeding himself; the tube wound receiving appropriate care.

About this time I again had the opportunity of examining him; found his lesions in process of repair, some entirely healed. He was advised to try swallowing his food to determine if he could do so, if it was painless, and if it was practical. At first it was difficult because of non use of the pharynx for so many months and the partial loss of the epiglottis, but he finally succeeded, and much to his delight, without pain. He soon was able to swallow solid foods, but liquids, especially water, still gave him difficulty. He became so enthusiastic that he overdid it, and his laryngeal lesions became active again.

A recent examination, about one year after his gastrostomy, shows a large ulcer of the whole of the larynx and portions of the pharynx, with loss of the epiglottis and marked edema of the tissue adjacent to ulceration. Even so extensive an involvement is not painful.

Recently casts and albumen have appeared, with edema of the legs and face. I was apprised of his death about February first.

This patient lived about sixteen months in comparative comfort because of his gastrostomy.

Case 3. Also a patient at the National Jewish Hospital, did not turn out so well. It illustrates the necessity of operating, if such is contemplated, before too much general exhaustion has occurred and the advisability of selecting the cases. I did not have the pleasure of seeing this case at all.

N. M., male, 30 years of age, Jewish, tailor by occupation. Family history: Negative.

Personal history. Usual diseases of childhood; frequent colds; tonsil operation two years ago.

Present illness began two years ago with a slight temperature, cough, expectoration and progressive weakness with loss of weight.

Examination. He was 5 feet, 6 inches in height; weight 114 pounds emaciated and anemic looking.

Hemoglobin 80%; red blood count 4,500,000; white blood count 9,800; blood Wassermann negative. Urine showed pus cells and some hyaline and granular casts; sputum positive to tubercle bacillus.

The X-ray of chest showed a far advanced tuberculosis with inflammatory mottling and fibrosis throughout both lungs, multiple cavities in upper left lobe, drop heart, adhesions to right diaphragm.

The throat showed extensive tuberculosis of larynx, with edema and beginning ulceration of the epiglottis. There was great dysphagia, and swallowing became so painful that he desisted and began a period of partial starvation.

He requested a gastrostomy which was reluctantly granted and performed. He stood the operation well and improved for a few days; then rapidly declined, ran a temperature to 104°, which previously ranged from 97° to 101°; became semi-delirious and died thirteen days after the operation.

Case 4. Patient in tuberculosis department, City and County Hospital. F. F., White, Male, 35 years of age, single, waiter. Family history, Negative. No tuberculosis in family.

Past History. Usual diseases of childhood. General health good until 20 years of age. Always susceptible to colds and sore throat.

Present illness began in 1908, with a slow onset. He developed a dry cough, slight expectoration, loss of weight, strength and appetite. Complained of hoarseness, pleurisy, shortness of breath and hemoptosis. Pneumonia in 1912. He now has periods of severe cough, lasting one-half hour, especially in mornings, evenings, and while eating. Great dysphagia and dyspnoea. Onset of laryngeal symptoms in April, 1920, consisting of dysphagia, dryness, dysphonia. These all advanced unfavorably.

Laboratory findings. Sputum positive to bacillus tuberculosis; urine negative. X-ray, extensive fibrosis upper half of left chest; considerable fibrosis in right apex and hylus; some mottling of right lung indicative of activity and cavitation of both apices. (Crosby)

Physical Examination. He is cachetic and poorly nourished.

Chest. Third stage tuberculosis, bilateral cavitation.

Neck. Cervical adenopathy.

Larynx. An hypertrophic infiltrative tuberculosis, with ulceration of epiglottis and ventricular bands. Much dysphagia.

Neck. ervical adenopathy.

Hand. Lupus.

Treatment. Silence and five per cent. cocain spray for pain.

This case was gastrostomized February 10th, 1922, on account of severe dysphagia and a progressive decline.

During March, 1922, he left the City and County Hospital, entering the Jewish Consumptives' Relief Society Sanatorium, where he died April 8th, 1922, from a terminal pulmonary edema.

This institution very courteously favored me with a report of his progress, which is added to complete the case, even though it did not form a part of the paper as originally read.

"Admitted to our institution March 21st, 1922. Temperature fluctuating between 100° and 103° daily, pulse 120, respiration 30. Extreme emaciation, slight burning pain in throat.

Pulmonary lesion: Extensive fibro-ulcerative process throughout left lung and right upper lobe with cavitation of left upper lobe.

Food introduced through gastrostomy tube would escape through sides of wound. To correct this, tube was transfixed to avoid seepage. But only about one ounce of liquid could be introduced. An effort to introduce more than that amount would result in a gurgling back of the fluid through the tube.

We were anxious to determine the exact size of the stomach by means of the X-ray but his marked general debility made it prohibitive. He had a severe craving for narcotics, and we suspected him of being a confirmed morphine habitue. Death occurred on April 8th, 1922, from a terminal pulmonary edema.

During the seven days prior to his death he had no laryngeal pain, but was markedly restless. Only a little less than one-half of one ounce of liquid could be introduced into the stomach and it was necessary to wait more than thirty minutes for another feeding without noticing regurgitation through the tube.

The febrile course was not changed."

The synopsis of another case occuring at the Jewish Consumptives' Relief Society is also added.

Case 5. Dr. W. S. D., age 30, had a gastrostomy performed by Doctor Roe on December 28th, 1920, for severe dysphagia as a result of extensive destruction of the epiglottis and arytenoids. A general tuberculous bronchopneumonia followed, although the operation was performed under local anesthesia.

The difficulty of feeding through the stomach tube (contracted stomach) was the same, and apparently for the same cause, as in the case of Case 4, but there was complete freedom from laryngeal pain.

Death occurred on February 7th, 1921.

Discussion.

It must be remembered that extensive ulceration usually, but not always, occurs in the late stages of pulmonary tuberculosis. Occasionally we find it associated with mild, well localized pulmonary lesions. It is in these latter that most can be expected from gastrostomy.

Cases should therefore be selected, first as to cure of the laryngeal lesions; and second as to their relief. In the latter the patient should be informed of what to expect.

Death from starvation and thirst must be a terrifying experience and in the absence of other effectual measures to relieve it, gastrostomy should be performed, even though no reason other than relief of the starvation and thirst exists. The same justification holds for this, as for tracheotomy in laryngeal carcinoma to prevent death from suffocation.

Benefits: These may be summarized as,

(a) Rest to the larynx. It is needless to detail the advantages of rest.

(b) Better nutrition, which is evident. Tube feeding is certainly more effective than per oram over an ulcer, when the patient cannot or refuses to swallow on account of the pain.

(c) Relief of pain.

(d) Promotion of healing, which theoretically is tenable because of the rest and absence of food irritation and which has actually occurred in two of the cases.

(e) Diminution of the toxemia.

Objections:

(1) Surgical risk. If gastrostomy is to be done at all one should not wait until starvation has made a derelict out of the patient. It should be done earlier than that, and from the few

cases here reported, the patients have stood the operation quite well. General anesthesia should of course be avoided.

(2) Gastric Tube Feeding. The patient can probably answer this better than the physician. I believe that the patient would insist that the annoyance of tube feeding is far less than the difficulty and pain of per oram feeding. The contraction of the stomach, which occured in cases 4 and 5, though a perplexing and annoying complication; one requiring much patience and attention, should not be considered of sufficient importance to prohibit a gastrostomy and its benefits.

Do the results justify the procedure? From the few cases presented they do. In addition to the relief of the distressing symptoms there is lessening of the toxemia, probably due to increased nutrition; healing of the local lesions, and general pain in laryngeal tuberculosis.

It should be noted that after gastrostomy, local remedies e. g., galvano-cautery, stimulation of indolent ulcerations, removal of greatly diseased tissue thus promoting regeneration, soothing applications to acutely active lesions, etc., should not be abandoned.

The literature on this subject is very meagre. Lowen mentions that he found none and Freed had a similar experience. It is probably not a new procedure, but certainly has not received much consideration by laryngologists or surgeons. A good deal has been written about gastrostomy for relief of esophageal obstruction, but we have little pertaining to its use for relief of pain in laryngeal tuberculosis.

I wish to acknowledge my indebtedness to Doctor Freed, of the National Jewish Hospital, who reported the two cases occuring there and reviewed the case of Lowen in a paper read before the Sanatorium Association. From his manuscript I have taken much record data of cases 2 and 3. Also to Doctor I. D. Bronfin, who kindly furnished the synopsis of case 5 and the report of case 4 after it passed from my observation.

DISCUSSION.

Dr. F. R. Spencer, Denver, Colo.: I told Dr. Cooper a little while ago, before he got up to read his paper, that I felt I knew very little about this subject. It certainly is comparatively a new one. At first, the operation impressed me as a very radical one, and I think at first that it does seem quite radical. Perhaps the only reason we feel justified in advising it at all is because these patients are in very desperate straits. I do not mean by this that I want to disagree entirely with Dr. Cooper's suggestion that we want to advise

gastrostomy, but as I said such patients are in such desperate straits that they are grasping at straws, and they are apt to accept most anything. If we can get together more data; for instance, if we can have a larger series of cases,—and Dr Cooper has done the right thing in collecting data concerning these cases—we would be better able to tell what the future for this operation will be.

Dr. Gilbert, of Boulder, whom many of you know, and by the way one of our best internists in the State, telephoned me the other day and wanted to know what we thought of advocating a gastrostomy in a case which was becoming very desperate. This is a case he had sent to him very lately, with very extensive laryngeal involvement. I think he had examined the patient a few days before, and I had to tell him I knew very little about it. He said on one occasion when he was at Saranac Lake they had one or two patients who had been greatly benefited; the patients had not been cured, and one of them had died a few months following the operation but during the subsequent months they had been more comfortable and felt very grateful for the relief which they had been afforded during that time. But that does not excuse us for not looking for other methods of relief for these patients, especially since we all occasionally see very late cases, as I believe Dr. Cooper has emphasized. For instance, tracheotomy has not been very satisfactory in these cases, because they die no matter what we do for them, when we see them so late. Dr. Freudenthal, a year ago last June, mentioned the use of orthoform in late cases, and spoke of the use of an emulsion of orthoform, which may be of help in relieving the patient, although most everything we use is disappointing.

One thing which Dr. Levy mentioned yesterday in showing a large series of laryngeal cases, and several tuberculous cases,—is the fact that many Eastern physicians, and some of our very ablest ones, do not believe in treating these cases at all. In fact, they have condemned many of us for advocating the treatment of laryngeal tuberculosis. I do not think there is any question but what we can get our best results if we see our patients early. We have some cases that are cured, and the only reason we cured them is because we saw them early and treated them.

Dr. Mullin has demonstrated some of his X-ray work in the Army. This is only to emphasize the importance of early recognition and early treatment, so that perhaps we will not have to do gastrostomy. Amputation of the epiglottis is a radical measure and one which is rather disappointing. Disappointment is a thing we may have to face in doing a gastrostomy, so that it should be borne in mind.

I think Dr. Cooper has been quite wise in letting the patients decide whether they wanted this operation performed, or whether they wanted to let the disease take its course. I think he also is wise in advocating that this be done, if it be done at all, before the patients have lost so much weight from starvation, and they starve very rapidly during the last few days.

We recently had a case in which we might have considered gastrostomy, a man sixty-two years of age, with an old fibroid pulmonary tuberculosis, from which he might have recovered. When he was first examined he had a very extensive involvement of the larynx and beginning ulceration. The ulceration progressed very rapidly, so that he was unable to swallow, and everything that was done for him was of little or no avail.

I hope those of you who have had an opportunity to see a large number of these cases will be able to follow up gastrostomy and give us a large series of cases so that we may have definite knowledge concerning this in the future.

Dr. W. J. Mullin, Colorado Springs: This is a perfectly logical procedure, and I think I shall advise it in a certain type of cases. I do not quite take the view that Dr. Spencer does, that it is a formidable procedure, it can be done under local anesthesia, and anything I think, that saves these people suffering in their last days is a God-send to them. I find these patients can often take food better than they can take liquids, and especially is it difficult for them to take water, and when their fluid intake is cut down, they fail very rapidly, as the general condition suffers more for lack of fluids than it does for lack of food. As Dr. Cooper has said, it ultimately comes to a point where cocain, orthoform, and practically nothing else will relieve them of their pain, and when you give them morphine it seems to unnecessarily prolong their life.

I have a woman now, thirty-six years old, with carcinoma at the lower end of the esophagus, about six centimeters from the cardia. This is a case that is hopeless, and I advised gastrostomy, and she is going to live a few days in peace at least. It is surprising the wonderful peace of mind that that woman has had in the past ten days since she has had her gastrostomy. Before that, I would walk in the room and the expression on her face was very discouraging, as it was extremely painful for her to swallow.

While we know of course, that usually the laryngeal symptoms are in direct proportion to the amount of lung involvement, and are secondary to the pulmonary condition, still we do occasionally see cases where the laryngeal symptoms predominate, and if these patients are properly nourished, and if we could get the nourishment into them without causing them the terrific distress of swallowing it, we would expect results. It seems to me that it is in just this type of case that we would expect the best results from a gastrostomy. As far as it being a curative procedure, Dr. Cooper has brought out the point that it has been tried in only a small number of cases. By recommending it and using it in a larger series of cases, one may get an "arrest" of laryngeal conditions in a certain number of cases, and even if you get one result in a large number of failures, it would still be worth while, as it makes them more comfortable and there is very little out-look for them unless they are relieved in some way.

Dr. Wm. C. Bane, Denver, Colo.: Mr. Chairman, I think we are indeed fortunate in having presented four of the cases which the Doctor has seen and treated and has studied. Personally, I have no experience, having seen none of the cases, and consequently I do not feel that I can add anything which would be of any value whatever, except to encourage the work which Dr. Cooper has undertaken for the relief of these patients and to prolong their lives, because we know the suffering is very great, and he has stated in his report that very marked relief was rendered for some months.

Dr. Cooper (Closing): Mr. Chairman, I think the whole question is in embryo. Its chief advantage lies in the fact that you give rest to the larynx, short-circuit your food route, and the ulcerated regions are no longer subject to irritation. The muscular effort necessary to pass food by an obstructed and sore larynx is done away with. Another advantage is, the patients get food and the toxemia is lessened. The patients, who have had it, like it, and I think that in the course of the next year or two we will have enough cases and enough study to really give the matter a more definite standing in the treatment of laryngeal tuberculosis.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, Inc.

MINUTES.

The Twenty-eighth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., convened at the New Willard Hotel, Washington, D. C., May 4th, 5th and 6th, 1922.

The Society was called to order by the President, Dr. Lewis A. Coffin of Washington, D. C.

The following members registered during the sessions:

DR. WILLIAM J. ABBOTT.....Cleveland, Ohio
DR. JOHN H. ALLEN.....Portland, Me.
DR. F. C. ARD.....Plainfield, N. J.
DR. HUBERT ARROWSMITH.....Brooklyn, N. Y.
DR. JAMES A. BABBITT.....Philadelphia, Pa.
DR. WILLIAM C. BANE.....Denver, Colo.
DR. WILLIAM P. BARNDOLLAR.....Pittsburgh, Pa.
DR. JOSEPH C. BECK.....Chicago, Ill.
DR. F. NOLTON BIGELOW.....Providence, R. I.
DR. H. S. BIRKETT.....Montreal, Canada
DR. HUGH B. BLACKWELL.....New York, N. Y.
DR. WESLEY C. BOWERS.....New York, N. Y.
DR. W. C. BRAISLIN.....Brooklyn, N. Y.
DR. ALFRED BRAUN.....New York, N. Y.
DR. H. H. BRIGGS.....Asheville, N. C.
DR. JOHN E. BROWN.....Columbus, Ohio
DR. SETH A. BRUMM.....Philadelphia, Pa.
DR. W. SOHIER BRYANT.....New York, N. Y.
DR. RALPH BUTLER.....Philadelphia, Pa.
DR. DON M. CAMPBELL.....Detroit, Mich.
DR. R. BISHOP CANFIELD.....Ann Arbor, Mich.
DR. WILLIAM P. CHAMBERLIN.....Cleveland, Ohio
DR. WILLIAM E. CHENERY.....Boston, Mass.
DR. GEORGE M. COATES.....Philadelphia, Pa.
DR. LEWIS A. COFFIN.....New York, N. Y.
DR. LEE COHEN.....Baltimore, Md.
DR. E. N. COLVER.....Battle Creek, Mich
DR. THEODORE CORWIN.....Newark, N. Y.
DR. CLAUDE E. COOPER.....Denver, Colo.
DR. CHARLES N. COX.....Brooklyn, N. Y.

DR. ROBERT H. CRAIG.....Montreal, Canada
DR. JOHN F. CULP.....Harrisburg, Pa.
DR. FRANKLIN E. CUTLER.....Cleveland, Ohio
DR. EWING W. DAY.....Pittsburg, Pa.
DR. LEE WALLACE DEAN.....Iowa City, Iowa
DR. EDWARD B. DENCH.....New York, N. Y.
DR. JESSE W. DOWNEY, JR.....Baltimore, Md.
DR. JAMES GARFIELD DWYER.....New York, N. Y.
DR. WELLS P. EAGLETON.....Newark, N. J.
DR. FRANCIS P. EMERSON.....Boston, Mass.
DR. LINN EMERSON.....Orange, N. J.
DR. CURTIS C. EVES.....Philadelphia, Pa.
DR. JOHN F. FAIRBAIRN.....Buffalo, N. Y.
DR. THOMAS H. FARRELL.....Utica, N. Y.
DR. CALVIN A. FAUNCE, JR.....Boston, Mass.
DR. LEWIS FISHER.....Philadelphia, Pa.
DR. HENRY HALL FORBES.....New York, N. Y.
DR. EDMOND P. FOWLER.....New York, N. Y.
DR. T. E. FULLER.....Texarkana, Ark.
DR. ISIDORE FRIESNER.....New York, N. Y.
DR. PERRY G. GOLDSMITH.....Toronto, Canada
DR. MAX A. GOLDSTEIN.....St. Louis, Mo.
DR. JAS. S. GREENE.....New York, N. Y.
DR. JOSEPH B. GREENE.....Asheville, N. C.
DR. LOUIS K. GUGGENHEIM.....St. Louis, Mo.
DR. J. H. GUNTZER.....New York, N. Y.
DR. THOMAS J. HARRIS.....New York, N. Y.
DR. WILLIAM G. HARRISON.....Birmingham, Ala.
DR. WILLIAM H. HASKIN.....New York, N. Y.
DR. HAROLD HAYS.....New York, N. Y.
DR. HALSTEAD S. HEDGES.....Charlottesville, Pa.
DR. I. M. HELLER.....New York, N. Y.
DR. F. T. HILL.....Waterville, Me.
DR. HAMPTON P. HOWELL.....New York, N. Y.
DR. E. V. HUBBARD.....New York, N. Y.
DR. THOMAS HUBBARD.....Cleveland, Ohio
DR. LESTER MEAD HUBBY.....New York, N. Y.
DR. SAMUEL IGLAUER.....Cincinnati, Ohio
DR. JOHN M. INGERSOLL.....Cleveland, Ohio
DR. H. J. INGLIS.....Boston, Mass.
DR. J. W. JERVEY.....Greenville, S. C.
DR. CLARENCE PORTER JONES.....Newport News, Va.
DR. CHARLES C. JONES.....Cincinnati, Ohio
DR. J. C. KEELER.....Philadelphia, Pa.
DR. GEORGE F. KEIPER.....Lafayette, Ind.
DR. J. S. KIRKENDALL.....Ithaca, N. Y.
DR. FRANK E. KITTREDGE.....Nashua, N. H.
DR. DANIEL W. LAYMAN.....Indianapolis, Ind.
DR. FIELDING O. LEWIS.....Philadelphia, Pa.
DR. H. I. LILLIE.....Rochester, Minn.

DR. H. W. LOEB.....St. Louis, Mo.
DR. JAMES E. LOGAN.....Kansas City, Mo
DR. ROBERT CLYDE LYNCH.....New Orleans, La.
DR. JOHN EDMUND MACKENTY.....New York, N. Y.
DR. GEORGE W. MACKENZIE.....Philadelphia, Pa.
DR. A. A. MACLACHLAN.....Pittsburgh, Pa.
DR. PERCY E. D. MALCOLM.....New York, N. Y.
DR. GEORGE PAULL MARQUIS.....Chicago, Ill.
DR. WILLIAM BEVERLY MASON.....Washington, D. C.
DR. JUSTUS MATTHEWS.....Minneapolis, Minn.
DR. JAMES F. McCAW.....Watertown, N. Y.
DR. JAMES HOMER McCREADY.....Pittsburg, Pa.
DR. SAMUEL McCULLAGH.....New York, N. Y.
DR. RICHMOND McKINNEY.....Memphis, Tenn.
DR D. McPHERSON.....New York, N. Y.
DR. CHAS. S. MEANS.....Columbus, Ohio
DR. WILLIAM F. MERCER.....Richmond, Va.
DR. LEONIDAS L. MIAL.....Morristown, N. J.
DR. CLIFTON M. MILLER.....Richmond, Va.
DR. ROBERT MILLIGAN.....Pittsburgh, Pa.
DR. THOMAS W. MOORE.....Huntington, W. Va.
DR. ROBERT L. MOORHEAD.....Brooklyn, N. Y.
DR. HARRIS P. MOSHER.....Boston, Mass.
DR. WILLIAM V. MULLIN.....Colorado Springs, Colo.
DR. ROBERT C. MYLES.....New York, N. Y.
DR. WALTER H. NARDIN.....Anderson, S. C.
DR. THEODORE E. OERTEL.....Augusta, Ga.
DR. LAFAYETTE PAGE.....Indianapolis, Ind.
DR. WENDELL C. PHILLIPS.....New York, N. Y.
DR. NORVAL H. PIERCE.....Chicago, Ill.
DR. JOHN B. RAE.....New York, N. Y.
DR. CHARLES W. RICHARDSON.....Washington, D. C.
DR. WALTER ROBERTS.....Philadelphia, Pa.
DR. JOHN A. ROBINSON.....New York, N. Y.
DR. SYLVAN ROSENHEIM.....Baltimore, Md.
DR. DUNBAR ROY.....Atlanta, Ga.
DR. C. C. SANDELS.....Pittsburgh, Pa.
DR. L. A. SCHIPFER.....Bismarck, No. Dak.
DR. G. O. SHARRETT.....Cumberland, Md.
DR. GEORGE E. SHAMBAUGH.....Chicago, Ill.
DR. B. R. SHURLY.....Detroit, Mich.
DR. JOHN R. SIMPSON.....Pittsburgh, Pa.
DR. ROSS HALL SKILLERN.....Philadelphia, Pa.
DR. GREENFIELD SLUDER.....St. Louis, Mo.
DR. HERBERT E. SMYTH.....Bridgeport, Conn.
DR. ROBERT SONNENSCHEIN.....Chicago, Ill.
DR. FRANK R. SPENCER.....Boulder, Colo.
DR. FREDERICK N. SPERRY.....New Haven, Conn.
DR. EDGAR H. STEIN.....Lancaster, Pa.
DR. OTTO JACOB STEIN.....Chicago, Ill.

DR. WALDO CHARLES STICKLE.....Brooklyn, N. Y.
DR. J. A. STUCKY.....Lexington, Ky.
DR. JOHN J. SULLIVAN, JR.....Scranton, Pa.
DR. EUGENE L. VANSANT.....Philadelphia, Pa.
DR. ANTONIE P. VOISLAWSKY.....New York, N. Y.
DR. D. H. WALKER.....Boston, Mass.
DR. N. S. WEINBERGER.....Sayre, Pa.
DR. WALTER H. WELLS.....Washington, D. C.
DR. JOSEPH A. WHITE.....Richmond, Va.
DR. LEON E. WHITE.....Boston, Mass.
DR. HAROLD WILSON.....Detroit, Mich.
DR. THOMAS C. WORTHINGTON.....Baltimore, Md.
DR. SIDNEY YANKAUER.....New York, N. Y.

EXECUTIVE SESSION.

Order of Business:

The Chairman extended a welcome to any guests who might be present, and invited them to take part in discussions.

Election of Members:

Dr. H. L. Baum,	Metropolitan Bldg., Denver, Colo.
Dr. Roy A. Barlow,	Farmers' Trust Bldg., South Bend, Ind.
Dr. Gordon Berry,	19 Elm St., Worcester, Mass.
Dr. Louis E. Brown,	Second National Bank Bldg., Akron, O.
Dr. Frank A. Burton,	Watts Bldg., San Diego, Cal.
Dr. C. C. Charlton,	124 So. Illinois Ave., Atlantic City, N. J.
Dr. Edwin Cobb,	Marshalltown, Ia.
Dr. J. K. M. Dickie	196 Elgin St., Ottawa, Can.
Dr. T. R. Gittins,	Davidson Bldg., Sioux City, Ia.
Dr. G. F. Harkness,	Putnam Bldg., Davenport, Ia.
Dr. D. C. Jarvis,	Howland Bldg., Barre, Vt.
Dr. Louis Levy,	Bank of Commerce Bldg., Memphis, Tenn.
Dr. A. G. Morris,	303 Alexander St., Rochester, N. Y.
Dr. C. T. Porter,	520 Commonwealth Ave., Boston, Mass.
Dr. C. M. Sautter,	11 E. 48th St., New York City.
Dr. E. C. Sewall,	3515 Pacific Ave., San Francisco, Cal.
Dr. E. M. Seydell,	201 No. Main St., Wichita, Kan.
Dr. J. J. Shea,	Exchange Bldg., Memphis, Tenn.
Dr. W. M. Simpson,	Memphis, Tenn.
Dr. J. F. Strauss,	104 So. Michigan Ave., Chicago, Ill.
Dr. H. G. Tobey,	416 Marlboro St., Boston, Mass.

Dr. Harris moved that the Secretary cast one ballot for the election of these members. This motion was seconded, and carried. The Secretary announced that one ballot was cast and the election carried.

The Nominating Committee consisted of the following names: Drs. J. A. Stucky, W. B. Chamberlin, N. H. Pierce, H. W. Loeb, F. P. Emerson.

It was moved and seconded that an Auditing Committee be appointed by the Chair. The Chairman appointed Drs. Dean, Dench

and Arrowsmith to serve as an Auditing Committee, to report at the next Executive Session.

REPORTS OF SECTION MEETINGS.

Report of Eastern Section:

Dr. J. F. Fairbairn reported for the Eastern Section that the meeting was held at the Buffalo General Hospital, Feb. 11, 1922. An important resolution was passed by the Council, requesting that a committee of three be appointed to confer with a similar committee of the American Neurological Association in regard to the results of the Barany tests, and to report thereon at the next meeting of the Triological Society.

Report of Southern Section:

Dr. T. C. Worthington reported for the Southern Section, that the meeting at Baltimore in January, although hampered by a severe snow storm, had managed to secure a valuable clinical meeting, and it was desired to thank the members for attending in spite of difficulties.

Report of Mid-Western Section:

I beg to report in regard to the Mid-Western Section of the Society, which met in Denver, on the afternoon of February 14th, and the entire day of February 15th. While the membership in this district is limited, only twelve or fourteen men being members, we had a registered attendance of fifty-two men doing this special work, and also nine of our members were present and registered.

The afternoon of February 14th, was devoted to clinics given by different members of our profession in Denver, several by the members of this Society and others by invited guests. These seemed to excite a good deal of interest and were greatly appreciated by the doctors present. The impression seemed to prevail that it was an innovation that was well worth while. And an expression of satisfaction at the demonstrations as well as the operative work that was presented was quite universal.

The scientific meeting of the Society took place on Wednesday morning, February 15th, at 9 o'clock, and continued with the papers and discussions until lunch time. In the afternoon again had the balance of the papers, eight in all for the day, which excited a good deal of discussion and seemed to be interesting in every respect. From the papers presented by the invited guests and also the discussions that were brought out, demonstrated to members of the

Society that we had too few members among this number of excellent men, and that an effort should be made to have more members in this section of the country, which is exceedingly large in extent, and has a great many men who are practicing scientifically and well this specialty. At the present time on account of the limited number of members in this section, it seems that the only feasible point for this meeting is in Colorado, either in Pueblo, Colorado Springs or in Denver; or in one of the smaller cities where we have at least one member. As the Chairmanship in this section, as well as in other Sections involves a great deal of attention and time of the one who is elected for a meeting, it confines the work to the territory where the most members are residents or where it is most convenient to reach to attend these meetings. The Chairman personally feels under many obligations to the men in this Section who aided most splendidly in an effort to get up this meeting which we all felt was quite successful, I am,

Yours most sincerely,

JOHN M. FOSTER.

Report of Middle Section:

The Middle Section of this Society was held at the University Club, St. Louis, Mo., February 22, 1922, and the following program presented:

1. Remarks by the Chairman.
   Greenfield Sluder ..... St. Louis
2. The Damping Mechanism of the Internal Ear.
   A. G. Pohlman—(By invitation) ..... St. Louis
3. Actinomycosis of the Tongue.
   G. B. New ..... Rochester, Minn.
4. The Radical Cure of Trigeminal Neuralgia.
   W. P. Coughlin—(By invitation) ..... St. Louis
5. X-ray Diagnosis of Unusual Esophageal Cases. Lantern Slides.
   Samuel Iglauer ..... Cincinnati
6. End Results of the Wiener-Sauer Lacrymal Sac Operation.
   W. E. Sauer ..... St. Louis
7. Fatalities Following Minor Operations on the Nose and Throat not Resulting from Anesthesia. Preliminary Report.
   H. W. Loeb ..... St. Louis

8. Some Remarks on the Borderline Cases between Otolaryngology and Neurology from the Viewpoint of a Neurological Surgeon.
   Ernest Sacks—(By invitation).....St. Louis
9. External Nasal Reconstruction.
   V. P. Blair—(By invitation).....St. Louis

GREENFIELD SLUDER,
Chairman.

Report of Western Section:

The Western Section Meeting of this Society was held at the rooms of the San Francisco County Medical Society, San Francisco County Medical Society, San Francisco, Cal., February 23rd and 24th, 1922 and the following program was presented.

1. The Tonus Effects of the Cristae and Maculae.
   by Professor S. S. Maxwell (Dept. Physiology U. of C.)
2. Studies of Hay Fever Producing Plants in the Western States. Demonstration of lantern slides and mounted Botanical Specimens.
   by Professor H. M. Hall (Carnegie Foundation)
3. Value of Vaccines in Hay Fever (true and false)
   by Albert Rowe, M. D., (Internist)
4. Bronchoscopic Removal of Foreign Bodies from the Lungs with Report of Three Recent Cases and Discussion of Difficulties.
   by Hill Hastings, M. D., Los Angeles, Cal.
5. Certain Surgical Considerations Underlying Acute Mastoid Surgery.
   by Eugene R. Lewis, M. D., Los Angeles, Cal.
6. A Demonstration of a Case Presenting Difficulties in the Differential Diagnosis of Brain Tumor vs. Hysteria.
   by E. C. Sewall, M. D., San Francisco, Cal.
7. Exhibition of Cases of Carcinoma of Nasal Sinuses Treated by Surgical Methods and Radium.
   by H. B. Graham, M. D., San Francisco, Cal.

GRANT SELFRIDGE,
Chairman.

SCIENTIFIC SESSION.

Exhibition of Instruments:

Dr. Perry Goldsmith showed a simple apparatus designed for autoinjection of the larynx, for the patient's use. The distal end con-

sisted of a rubber catheter-like tube, passed into the nose. This communicated, by means of a U-shaped steel tube, with a bellows. The mouth is kept closed, and with pressure of the bellows, abundant fluid is forced through the passages. The psychic effect was encouraging to the patient, who, apart from the consideration of medication, felt he was enabled to do something for himself.

Dr. Haskin presented two plates illustrating, first, the passage of the optic nerve through the sphenoid, in a canal half an inch long, and clearly surrounded by the wall of the sphenoid; second, X-rays of six infantile temporal bones.

Two communications were then read: one from the U. S. P. H. Service, in regard to the National Museum medical collection; the other in regard to the work of the Volta Bureau, an institution doing useful work in propaganda for the help of deaf people and deserving of the support of the Otologists.

Dr. Wendell Phillips urged the members to take advantage of the invitation of the Volta bureau, and to take a closer interest in the sociological side of the problem of deafness.

An announcement was then made in regard to the International Congress of Otology, in Paris, in July. Any members able to attend were asked to present their names, so that they might be duly accredited as delegates. Drs. Birkett, Dench, Eagleton, Walker, Forbes, Fairbairn, C. Jackson, Cutler and L. Levy were listed as those intending to be present.

EXECUTIVE SESSION.

May 5th, 1922.

Secretary's Report:

To the Fellows of the American Laryngological, Rhinological and Otological Society, Incorporated:

Gentlemen:

The past year has brought a great loss to the Society in the deaths of Dr. H. L. Lynah, Dr. D. A. Kuyk and Dr. H. Bert Ellis.

We have had only one resignation, that of Dr. J. A. Winslow, but have lost three members because of non-attendance.

Twenty-one new members were elected Active Fellows and we have thirty-six approved applications awaiting final approval of their theses to be submitted on November 1st, 1922.

Respectfully submitted,

W. H. Haskin,
Secretary.

Treasurer's Report.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL and OTOLOGICAL SOCIETY, STATEMENT OF RECEIPTS AND DISBURSEMENTS MAY 23, 1921 TO APRIL 24, 1922.

Balance May 23, 1921		$219.75
Receipts.		
Transferred from Savings Bank	$900.00	
Received from First National Bank	1.20	
Dividends from Penna. R. R. stock	100.00	
Cresson School Bonds	180.00	
Dividends from Westinghouse Air Brake Co.	140.00	
Coupons	285.62	
1 Dues received year 1916-1917	10.00	
1 Dues received year 1919-1920	10.00	
23 Dues received year 1920-1921	230.00	
273 Dues received year 1921-1922	2,732.20	
15 Initiations	150.00	
		$ 4,739.02
		$ 4,958.77

Disbursements.

Vol. No.		
1	Mrs. V. B. Alvarez, part payment reporting annual meeting	$ 28.00
2	Cash, Postage stamps Treasurer's office	9.00
3	Alfred Moore, Lantern two days	40.00
4	Peoples Nat'l. Bank, Payment on loan	900.00
5	Dr. W. H. Haskin, Expenses Secretary 1920-21	200.00
6	Dr. D. J. G. Wishart, Expense publication, Training of the Specialist	10.00
7	Mrs. V. B. Alvarez, Reporting annual meeting	80.00
8	People's Nat'l Bank, Interest and loan	19.45
9	People's Nat'l Bank, Payment on loan and interest	602.40
10	L. V. Sattele, Auditing books 1920-21	7.50
11	Commonwealth Trust Co., Safe Deposit Box	5.00
12	Peoples Nat'l Bank, Exchange on Canadian check	2.93
13	Dr. Jos. B. Green, Reporting meeting Southern section	25.00
14	First Nat'l. Bank, Exchange on Canadian check	1.71
15	University Club New York, Council Dinner	84.00
16	Dr. Geo. L. Richards, Expenses Publication Committee for 1921-22	114.74
17	Mercury Publishing Co., Transactions, Printing, Postage, etc.	1,104.80
18	Dr. J. M. Foster, Reporting meeting Mid-West Section	19.60
19	Mercury Publishing Co., Postage, circulars, circular letters, letter heads	445.12
20	Dr. W. H. Haskin, Expenses Secretary 1921-22	200.00

21	Dr. Grant Selfridge, Reporting meeting Western Section	25.00	
22	Dr. E. W. Day, Expenses Treasurer 1921-22	75.00	
			$ 3,999.34
			$ 959.43
	Bank balance as shown by 1st Nat'l. Bank statement	984.43	
	Voucher No. 21 Dr. Grant Selfridge outstanding	25.00	
			$ 959.43

Above statement audited, and hereby certify that I have found the same to be correct.

COMMONWEALTH TRUST CO. OF PGH.,
W. M. SHERIDAN,
Secretary.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL and OTOLOGICAL SOCIETY, STATEMENT OF INVESTED ACCOUNTS AT THE CLOSE OF BUSINESS APRIL 24, 1922

Cash deposited with First National Bank, Pittsburgh, Pa., in Savings Account, pass book No. F71260, as follows:

May 23, 1921 Balance		$ 908.82
Receipts.		
June 1, 1921 Interest	$ 14.13	
June 7, 1921 Dividend P. R. R. stock	50.00	
July 26, 1921 Liberty Loan coupons	58.76	
July 30, 1921 Westinghouse Air Brake stock	70.00	
Mar. 3, 1922 Dividend P. R. R. stock	50.00	
Dec. 1, 1921 Interest, First Nat'l. Bank	2.96	
		$ 245.85
Total		$ 1,154.67
Disbursements.		
Transferred to Checking Account		900.00
Balance on hand April 24, 1922		$ 254.67

	Investments.	*Par Value.*	*Market Value.*	*Return.*
100	Shares Penna. R. R. Stock	$5,000.00	$4,200.00	$200.00
40	Shares Westinghouse Air Brake	2,000.00	3,600.00	160.00
8	Shares Cresson Borough School 4½s Bonds ($500 each)	4,000.00	4,000.00	180.00
2	1st L. L. 3½% Bonds	2,000.00	2,000.00	70.00
2	4th L. L. 4¼% Bonds	2,000.00	2,000.00	90.00
2	Victory 4¾% Bonds ($500 ea.)	1,000.00	1,000.00	47.50
1	United Kingdom Great Britain and Ireland Bond 5½s	1,000.00	1,070.00	55.00
1	Govt. of Norway 8% Bond	1,000.00	1,120.00	80.00
		$17,000.00	$17,990.00	$882.50

Statement audited and certified as being correct. Stocks and Bonds checked and all found to be on hand, also bonds having all the necessary coupons attached.

COMMONWEALTH TRUST CO. OF PGH.,
W. M. SHERIDAN,
Secretary.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL and OTOLOGICAL SOCIETY, STATEMENT OF RECEIPTS AND DISBURSEMENTS MAY 23, 1921 to April 24, 1922.

Cash on hand May 23, 1921		$ 219.75
Receipts.		
Transferred from Savings Account	$ 900.00	
Received from Investments	70.62	
Received from First National Bank (refund)	1.20	
Dues	2,982.20	
Initiations	150.00	
	$	4,739.02
Total		$ 4,958.77
Disbursements. (per detailed statement attached)		
Expenses Annual meeting	$ 148.00	
Expenses Section meetings	69.60	
Payment of note of 1921 with interest	1,521.85	
Expenses Council	94.00	
" Secretary 1921-1922	845.12	
" Treasurer	101.14	
" Publication Committee	1,219.63	
	$	3,999.34
Cash on hand April 24, 1922		$ 959.43

Statement of Investment Account.

Cash deposited with First National Bank, Pittsburgh, Pa., in Savings Account, pass book No. F 71260, as follows:

Cash on hand May 23, 1921		$ 908.82
Receipts from Investments.		
Interest	$ 17.09	
Dividends Penna. R. R. Stock	100.00	
Liberty Loan Coupons	58.76	
Dividends Westinghouse Air Brake Stock	70.00	
	$	245.85
Total		$ 1,154.67
Disbursements.		
Transfer to Checking Account		$ 900.00
Balance on hand April 24, 1922		$ 254.67

Report of Nominating Committee:

Board of Directors.

Drs. Dunbar Roy, F. Nolton Bigelow, Gordon B. New, W. G. Harrison, Robert Levy, Isaac H. Jones, William H. Haskin, Ewing W. Day, H. P. Mosher, J. F. McCaw, J. W. Jervey, L. W. Dean, J. F. McKernon, N. H. Pierce, L. A. Coffin, B. R. Shurley, Ross H. Skillern, George L. Richards.

Miscellaneous Business.

Dr. Shambaugh presented the matter of the publication of a new special journal on oto-laryngology, to be edited and financed by the A. M. A. The new premises of the A. M. A. are to be completed shortly which will allow of their undertaking the publication of a new special journal, in addition to the five already under publication.

Dr. Phillips, as a Trustee of the A. M. A., said that this publication would be undertaken, without idea of profit to the Association, as long as it paid expenses.

Dr. Day moved that the matter be referred to the Council with power. This was duly seconded and carried.

Dr. Haskin stated that the proposed Chairman of the Mid-Western Section was physically unable to handle the work, and asked that another man be nominated as substitute, as the Section was a very active one.

This motion was seconded and carried.

Dr. Phillips moved that at the end of the session the Society stand a moment in memory of the members who had passed on.

This was duly seconded and carried.

Dr. Loeb, on behalf of the Committee on Necrology read a report on Dr. Lynah, prepared by Dr. Arrowsmith.

The Chairman stated that the Society had lost other fine men, such as Dr. H. Burt Ellis, but that reports had not yet been prepared.

The Society then remained standing a minute in respect to the memory of those who had died during the past year.

This concluded the Business Session.

EXECUTIVE SESSION.

May 6th, 1922.

It was moved and seconded that a vote of thanks be presented to the Medical Society of the District of Columbia for their kindness in loaning the hall for the meeting. This was carried.

Dr. Mosher moved a vote of thanks be extended to Dr. and Mrs. Wells for their courteous entertainment of the Society. This was seconded and carried.

A vote of thanks and appreciation was extended to Dr. Richardson for his help in making arrangements for the hall. This was seconded and carried.

The following before the legislature was approved, and it was moved and seconded that the Society endorse the bill. Carried.

Whereas: There are some sixty ex-service men totally deaf. This disability being incurred while in the naval or military forces of this country during the late war and about one half this number are not receiving full compensation and many of these are physically unfit to take vocational training, or to hold jobs could they secure them.

Be it resolved by the American Laryngological, Rhinological and Otological Society, that it endorses and urges the passage of Senate Bill 1497 and House Bill 6422 which bills would allow full disability in these cases.

The election of Board of Directors then took place. The Secretary was instructed to cast one ballot for their election. This was done and the election closed.

A meeting of the newly elected Board of Directors was held in Washington, D. C., in the building of the District of Columbia Medical Society, on May 6th, 1922.

Dr. H. P. Mosher was elected temporary Chairman and Dr. W. H. Haskin, Secretary.

On motion duly seconded and carried the Secretary was instructed to cast one ballot for all officers as recommended by the Nominating Committee as follows:

President: Dunbar Roy.

Vice Presidents: Eastern Section, F. Nolton Bigelow; Southern Section, Wm. G. Harrison; Middle Section, Gordon B. New; Mid-Western Section, Robert Levy; Western Section, Isaac H. Jones.

Secretary: William H. Haskin.

Treasurer: Ewing W. Day.

Editor: G. L. Richards.

Class A: Drs. H. P. Mosher, J. F. McCaw and J. W. Jervey.

Class B: Drs. L. W. Dean, J. F. McKernon and N. H. Pierce.

Class C: Drs. L. A. Coffin, B. R. Shurley and R. H. Skillern.

The Secretary announced that he had cast the ballot and the officers were declared duly elected.

The meeting adjourned to meet on January 6th, 1923.

Present:— Drs. Dunbar Roy, F. N. Bigelow, I. H. Jones, R. H. Skillern, H. P. Mosher, E. W. Day and W. H. Haskin.

Respectfully submitted,

W. H. Haskin,

Secretary.

FELLOWS

1901 Dr. Jos. H. Abraham.....130 West 58th St., NewYork
1913 Dr. William J. Abbott..1836 Euclid Ave., Cleveland, Ohio
1915 Dr. Lawrence Dade Alexander, 130 West 59th St., New York
1908 Dr. John H. Allen.....711 Congress St., Portland, Me.
1905 Dr. Frank Allport.....7 West Madison St., Chicago, Ill.
1904 Dr. F. C. Ard.....604 Park Ave., Plainfield, N. J.
1903 Dr. Hubert Arrowsmith..170 Clinton St., Brooklyn, N. Y.
1899 Dr. William J. Atkinson...27 Church St., Paterson, N. J.
1905 Dr. Jas. A. Babbitt...2117 Chestnut St., Philadelphia, Pa.
1905 Dr. William C. Bane...Metropolitan Bldg., Denver, Colo.
1922 Dr. Roy A. Barlow, Farmer's Trust Bldg., South Bend, Ind.
1915 Dr. William P. Barndollar, Westinghouse Bldg., Pittsburg, Pa.
1904 Dr. John F. Barnhill....Penway Bldg., Indianapolis, Ind.
1911 Dr. Harry A. Barnes, 520 Commonwealth Ave., Boston Mass.
1922 Dr. H. L. Baum.....Metropolitan Bldg., Denver, Col.
1903 Dr. Joseph C. Beck.....108 N. State St., Chicago, Ill.
1895 Dr. T. Passmore Berens...25 Park Ave., New York City
1922 Dr. Gordon Berry.....19 Elm St., Worcester, Mass.
1918 Dr. F. Nolton Bigelow, 124 Waterman St., Providence, R. I.
1905 Dr. H. S. Birkett.....252 Mountain St., Montreal, Can.
1912 Dr. Hugh B. Blackwell.....148 West 58th St., New York
1906 Dr. Charles R. C. Borden, 520 Commonwealth Ave., Boston, Mass.
1918 Dr. Wesley C. Bowers..27 West 49th St., New York City
1898 Dr. W. C. Braislin.....425 Clinton Ave., Brooklyn, N. Y.
1915 Dr. Alfred Braun.....31 East 72nd St., New York
1914 Dr. H. H. Briggs.....73 Haywood St., Asheville, N. C.
1915 Dr. Clayton M. Brown..510 Delaware Ave., Buffalo, N. Y.
1911 Dr. H. Beattie Brown, 39 Baker St., Saranac Lake, N. Y.
1909 Dr. John E. Brown.....370 E. Town St., Columbus, Ohio
1916 Dr. John Mackenzie Brown, 1002 Brockman Bldg., Los Angeles, Cal.

1922 Dr. L. E. Brown...2nd National Bank Bldg., Akron, Ohio
1917 Dr. Seth A. Brumm....1411 Walnut St., Philadelphia, Pa.
1903 Dr. W. Sohier Bryant.....107 East 39th St., New York
1915 Dr. Louis J. Burns...1906 Chestnut St., Philadelphia, Pa.
1922 Dr. F. A. Burton.....Majestic Bldg., Denver, Col.
1914 Dr. Ralph Butler....1926 Chestnut St., Philadelphia, Pa.
1915 Dr. Don M. Campbell, 801 Peter Smith Bldg., Detroit, Mich.
1903 Dr. William E. Campbell, Atlanta National Bank Bldg., Atlanta, Ga.
1904 Dr. R. Bishop Canfield..330 S. State St., Ann Arbor, Mich.
1912 Dr. Thomas E. Carmody, Metropolitan Bldg., Denver, Colo.
1915 Dr. William Wesley Carter..2 West 67th St., New York
1908 Dr. William B. Chamberlin, 614 Osborn Bldg., Cleveland, Ohio.
1900 Dr. Talbot R. Chambers, 15 Exchange Pl., Jersey City, N. J.
1922 Dr. C. C. Charlton, 124 So. Illinois Ave., Atlantic City,N. J.
1900 Dr. William E. Chenery, 377 Commonwealth Ave., Boston, Mass.
1907 Dr. George M. Coates...1811 Spruce St., Philadelphia, Pa.
1922 Dr. Edwin Cobb.....Marshalltown, Ia.
1895 Dr. Lewis Coffin.....114 East 54th St., New York
1895 Dr. Henry Coggeshall.....40 East 58th St., New York
1914 Dr. Lee Cohen.....1820 Eutaw Pl., Baltimore, Md.
1899 Dr. Burnitt Collins, 645 St. Marks Ave., Brooklyn, N. Y.
1920 Dr. B. N. Colver, 182 Manchester St., Battle Creek, Mich.
1914 Dr. Ray Connor, 503 Washington Arcade, Detroit, Mich.
1900 Dr. Theodore Corwin.....20 Central Ave., Newark, N. J.
1916 Dr. Claude E. Cooper, Metropolitan Bldg., Denver, Colo.
1896 Dr. Charles N. Cox....257 Jefferson Ave., Brooklyn, N. Y.
1912 Dr. Gerhard H. Cox.....137 East 54th St., New York
1906 Dr. Robert H. Craig, 510 Sherbrooke St., W. Montreal, Can.
1897 Dr. Eugene A. Crockett, 298 Marlboro St., Boston, Mass.
1895 Dr. William Ledlie Culbert....16 East 54th St., New York
1904 Dr. John F. Culp.....410 No. Third St., Harrisburg, Pa.
1918 Dr. Henry Mortimer Cunningham, Birks Bldg., Vancouver, B. C.
1916 Dr. Franklin E. Cutler, 1025 Schofield Bldg., Cleveland, Ohio

1912 Dr. George E. Davis.....42 West 77th St., New York
1895 Dr. Ewing W. Day, Westinghouse Bldg., Pittsburgh, Pa.
1914 Dr. Lee Wallace Dean.....Iowa City, Iowa
1915 Dr. Frederick F. C. Demarest, 29 Academy St., Passaic, N. J.
1895 Dr. Edward B. Dench.....15 East 53rd St., New York
1918 Dr. Frank L. Dennis, Ferguson Bldg., Colorado Springs, Colo.
1922 Dr. J. K. M. Dickie.....196 Elgin St., Ottowa, Can.
1914 Dr. Jesse W. Downey, Jr., The Buckler, Charles and Center, Baltimore, Md.
1900 Dr. William H. Dudley, 512 Brockman Bldg., Los Angeles, Cal.
1898 Dr. Arthur B. Duel.....27 East 57th St., New York
1895 Dr. John Dunn....411 East Franklin St., Richmond, Va.
1920 Dr. Henry S. Dunning.....33 East 68th St., New York
1916 Dr. John W. Durkee.....142 Clinton St., Brooklyn, N. Y.
1921 Dr. Julius Dworetzky.....7 Law St., Liberty, N. Y.
1915 Dr. James Garfield Dwyer....40 East 41st St., New York
1900 Dr. Wells P. Eagleton....15 Lombardy St., Newark, N. J.
1916 Dr. Thomas O. Edgar, Dixon National Bank Bldg., Dixon, Ill.
1913 Dr. Joshua A. Ellegood, Equitable Bldg., Wilmington, Del.
1907 Dr. Francis P. Emerson, 520 Commonwealth Ave., Boston, Mass.
1907 Dr. Linn Emerson.....Metropolitan Bldg., Orange, N. J.
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1914 Dr. John F. Fairbairn...503 Delaware Ave., Buffalo, N. Y.
1900 Dr. Thomas H. Farrell.....236 Genesse St., Utica, N. Y.
1921 Dr. Calvin A. Faunce, Jr., 320 Commonwealth Ave., Boston, Mass.
1901 Dr. George Fetterolf, 2047 Chestnut St., Philadelphia, Pa.
1918 Dr. Lewis Fisher.....1820 Spruce St., Philadelphia, Pa.
1912 Dr. Mark O. Fisher.....Parkersburg, W. Va.
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1899 Dr. Ernest W. Fleming, 523 West 6th St., Los Angeles, Cal.
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1911 Dr. Edgar A. Forsyth.....471 Virginia St., Buffalo, N. Y.
1907 Dr. John M. Foster.....Metropolitan Bldg., Denver, Col.
1912 Dr. Edmond P. Fowler.....114 East 54th St., New York
1916 Dr. Ira Frank.....104 So. Michigan Ave., Chicago, Ill.

1901 Dr. Wolff Freudenthal.....24 West 88th St., New York
1921 Dr. T. E. Fuller, Texarkana National Bank Bldg., Texarkana, Ark.
1915 Dr. Isidore Friesner.....36 East 73rd St., New York
1922 Dr. T. R. Gittins.....Davidson Bldg., Sioux City, Ia.
1900 Dr. Thomas J. Gallaher...California Bldg., Denver, Colo.
1904 Dr. Perry G. Goldsmith....84 Carlton St., Toronto, Can.
1899 Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis, Mo.
1906 Dr. Charles Graef.....1125 Boston Rd., New York
1915 Dr. Harrington Bidwell Graham, Shreve Bldg., San Francisco, Cal.
1896 Dr. Charles P. Grayson, 262 South 16th St., Philadelphia, Pa.
1904 Dr. D. C. Greene, Jr.....23 Bay State Rd., Boston, Mass.
1919 Dr. Jas. S. Greene.....143 East 37th St., New York City
1913 Dr. Joseph Berry Greene, Haywood Bldg., Asheville, N. C.
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1916 Dr. Louis K. Guggenheim, 1006 Carleton Bldg., St. Louis, Mo.
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1895 Dr. Thomas H. Halsted, 405 Fayette Park, Syracuse, N. Y.
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1918 Dr. Isaac H. Jones....1920 Orange St., Los Angeles, Cal.
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1896 Dr. W. F. Knowles, 520 Commonwealth Ave., Boston, Mass.
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1908 Dr. Secord H. Large....536 Rose Bldg., Cleveland, Ohio
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1915 Dr. Daniel W. Layman, 608 Hume-Mansur Bldg., Indianapolis, Ind.
1896 Dr. M. D. Lederman.....58 East 75th St., New York
1904 Dr. George A. Leland, 354 Commonwealth Ave., Boston, Mass.
1922 Dr. Louis Levy, Bank of Commerce Bldg., Memphis, Tenn.
1895 Dr. Robert Levy.....Metropolitan Bldg., Denver, Colo.
1913 Dr. Eugene R. Lewis, 1920 Orange St., Los Angeles, Cal.
1915 Dr. Fielding O. Lewis..261 So. 17th St., Philadelphia, Pa.
1895 Dr. Robert Lewis.....48 West 40th St., New York
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1902 Dr. Chas. S. Means....137 East State St., Columbus, Ohio
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1911 Dr. William F. Mercer, 1101 West Franklin St., Richmond, Va.
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1911 Dr. Clifton M. Miller, Stuart Circle Hosp., Richmond, Va.
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1900 Dr. Gilbert D. Murray.....516 Spruce St., Scranton, Pa.
1901 Dr. Harry L. Myers, Taylor Bldg., 151 Granby St., Norfolk, Va.
1895 Dr. Robert C. Myles.....11 E. 48th St., New York
1915 Dr. Walter H. Nardin....Bleckley Bldg., Anderson, S. C.
1918 Dr. Gordon B. New..Mayo Foundation, Rochester, Minn.
1915 Dr. Theodore E. Oertel.....Lamar Bldg., Augusta, Ga.
1900 Dr. James P. O'Kelley, 501 Macheca Bldg., New Orleans, La.
1900 Dr. Seymour Oppenheimer..45 East 60th St., New York
1920 Dr. L. Ostrom....People's Bank Bldg., Rock Island, Ill.
1900 Dr. Francis R. Packard, 304 South 19th St., Philadelphia, Pa.
1908 Dr. John R. Page.....127 East 62nd St., New York
1900 Dr. Lafayette Page, 603 Hume Mansur Bldg., Indianapolis, Ind.
1902 Dr. J. Walter Park....32 North 2nd St., Harrisburg, Pa.
1903 Dr. Jas. A. Patterson, 214 Burns Building, Colo Springs, Colo.
1920 Dr. W. W. Pearson.....Equitable Bldg., Des Moines, Ia.
1895 Dr. Wendell C. Phillips.....40 West 47th St., New York

1896 Dr. Norval H. Pierce, 22 East Washington St., Chicago, Ill.
1922 Dr. Kaspar Pischell.....Butler Bldg., San Francisco, Cal.
1922 Dr. C. T. Porter, 520 Commonwealth Ave., Boston, Mass.
1899 Dr. John B. Rae.....247 West 70th St., New York
1920 Dr. Jas. E. Reeder.....Davidson Bldg., Sioux City, Ia.
1908 Dr. Andrew J. N. Reik, 1202 No. Charles St., Baltimore, Md.
1906 Dr. Henry O. Reik..300 East 30th Street, Baltimore, Md.
1895 Dr. W. Scott Renner..341 Linwood Ave., Buffalo, N. Y.
1898 Dr. George L. Richards, 124 Franklin St., Fall River, Mass.
1895 Dr. Charles W. Richardson, 1317 Connecticut Ave., Washington, D. C.
1905 Dr. Walter Roberts....1732 Spruce St., Philadelphia, Pa.
1901 Dr. W. Humes Roberts, 461 East Colo. St., Pasadena, Cal.
1907 Dr. John A. Robinson.....40 E. 41st St., New York City
1919 Dr. Charles M. Robertson, 30 N. Michigan Ave., Chicago, Ill.
1896 Dr. Arthur G. Root.....218 States St., Albany, N. Y.
1908 Dr. Sylvan Rosenheim..1710 Linden Ave., Baltimore, Md.
1896 Dr. George T .Ross.....456 Mackay St., Montreal, Can.
1895 Dr. Dunbar Roy, Grand Opera House Block, Atlanta, Ga.
1904 Dr. C. C. Sandels....Westinghouse Bldg., Pittsburgh, Pa.
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1922 Dr. C. M. Sautter.....11 East 48th St. New York City
1912 Dr. Rufus B. Scarlet, 78 North Clinton Ave., Trenton, N. J.
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1914 Dr. William A. Scruton, 30 Central Park South, New York City
1921 Dr. Grant Selfridge....Schroth Bdg., San Francisco, Cal.
1914 Dr. Eugene T. Senseney..308 Lister Bldg., St. Louis, Mo.
1922 Dr. E. C. Sewall....3515 Pacific Ave., San Francisco, Cal.
1922 Dr. E. M. Seydell.....201 No. Main St., Wichita, Kan.
1904 Dr. J. Clarence Sharp.....62 West 46th St., New York
1921 Dr. G. O. Sharrett.....45 Bedford St., Cumberland, Md.
1922 Dr. J. J. Shea.....Exchange Bldg., Memphis, Tenn.
1917 Dr. Geo. E. Shambaugh..People's Gas Bldg., Chicago, Ill.
1898 Dr. Warren S. Shattuck..160 Clinton St., Brooklyn, N. Y.
1907 Dr. B. R. Shurly....32 West Adams Ave., Detroit, Mich.
1915 Dr. John R. Simpson, Westinghouse Bldg., Pittsburgh, Pa.
1922 Dr. Wm. L. Simpson...Exchange Bldg., Memphis, Tenn.

1910 Dr. Ross Hall Skillern, 1928 Chestnut St. Philadelphia, Pa.
1911 Dr. Greenfield Sluder, 3542 Washington Ave., St. Louis, Mo.
1908 Dr. A. John Smith.....Yonkers, New York
1905 Dr. E. Terry Smith.....36 Pearl St., Hartford, Conn.
1903 Dr. Harmon Smith.....44 West 49th St., New York
1915 Dr. Owen Smith.....692 Congress St., Portland, Me.
1895 Dr. S. MacCuen Smith, 1429 Spruce St., Philadelphia, Pa.
1902 Dr. Herbert E. Smyth....376 John St., Bridgeport, Conn.
1921 Dr. Robert Sonnenschein, 4534 Michigan Ave., Chicago Ill.
1913 Dr. Frank R. Spencer..Physicians' Block, Boulder, Colo.
1911 Dr. Frederick N. Sperry, 33 College St., New Haven, Conn.
1906 Dr. Charles N. Spratt, Physicians' and Surgeons' Bldg., Minneapolis, Minn.
1904 Dr. George E. Steel.....256 W. 79th St., New York
1915 Dr. Edgar J. Stein.....134 N. Duke St., Lancaster, Pa.
1900 Dr. Otto Jacob Stein...30 North Michigan Ave., Chicago, Ill.
1915 Dr. Waldo Charles Stickle, 80 Pierrepont St., Brooklyn, N. Y.
1902 Dr. F. L. Stillman.....185 E. State St., Columbus, Ohio
1898 Dr. George C. Stout....1611 Walnut St., Philadelphia, Pa.
1922 Dr. J. F. Strauss....104 So. Michigan Ave., Chicago, Ill.
1895 Dr. J. A. Stucky..The McClelland Bldg., Lexington, Ky.
1915 Dr. John James Sullivan, Jr., 401 Traders Bank Bldg., Scranton, Pa.
1900 Dr. Clement F. Theisen, 172 Washington Ave., Albany, New York
1895 Dr. C. A. Thigpen....401 S. Court St., Montgomery, Ala.
1895 Dr. F. M. Thigpen....401 S. Court St., Montgomery, Ala.
1907 Dr. John J. Thompson....3 Park Ave., Mt. Vernon, N. Y.
1907 Dr. Wade Thrasher, N. E. corner 7th and Race Sts., Cincinnati, Ohio
1921 Dr. G. L. Tobey, Jr..416 Marlborough St., Boston, Mass.
1922 Dr. H. G. Tobey....416 Marlborough St., Boston, Mass.
1905 Dr. D. H. Trowbridge.....Fresno, Cal.
1906 Dr. Derrick T. Vail.....24 East 8th St., Cincinnati, Ohio
1915 Dr. Don Allison Vanderhoof, Exchange National Bank Bldg., Colorado Springs, Colo.

1896 Dr. Eugene L. Vansant, 2046 Chestnut St., Philadelphia, Pa.
1905 Dr. Antonie P. Voislawsky..33 East 68th St., New York
1915 Dr. Irving Wilson Voorhees, 114 East 54th St., New York
1896 Dr. Henry L. Wagner, 518 Sutter St., San Francisco, Cal.
1907 Dr. Ernest DeW. Wales, 1236 N. Pennsylvania St., Indianapolis, Ind.
1921 Dr. D. H. Walker, 390 Commonwealth Ave., Boston, Mass.
1900 Dr. James S. Waterman, 676 St. Marks Ave., Brooklyn, N. Y.
1914 Dr. Arthur I. Weil, 717 Maison Blanche Bldg. Annex, New Orleans, La.
1921 Dr. N. S. Weinberger, Robert Packer Hospital, Sayre, Pa.
1901 Dr. Walter A. Wells, The Rochambeau, Washington, D. C.
1920 Dr. Francis W. White, 174 West 58th St., New York City
1918 Dr. Leon E. White, 390 Commonwealth Ave., Boston, Mass.
1895 Dr. Joseph A. White, 200 East Franklin St., Richmond, Va.
1910 Dr. Frederick Whiting.....19 West 47th St., New York
1908 Dr. Alfred Wiener.....550 Park Ave., New York City
1916 Dr. Harold Wilson, 1501 David Whitney Bldg., Detroit, Mich.
1921 Dr. Gordon Wilson, 104 So. Michigan Ave., Chicago, Ill.
1896 Dr. Norton L. Wilson, 410 Westminister Ave., Elizabeth, N. J.
1900 Dr. D. J. Gibb Wishart..47 Grosvenor St., Toronto, Can.
1904 Dr. George B. Wood....1830 Spruce St., Philadelphia, Pa.
1898 Dr. John F. Woodward....153 Granby St., Norfolk, Va.
1909 Dr. Thomas C. Worthintgon, 1102 N. Charles St., Baltimore, Md.
1909 Dr. Sidney Yankauer.....616 Madison Ave., New York

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1897 Dr. F. C. Cobb.....11 Marlboro St., Boston, Mass.
1901 Dr. J. Solis Cohen..2113 Chestnut St., Philadelphia, Pa.
1895 Dr. Frank Hyatt....The Rochambeau, Washington, D. C.
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HONORARY FELLOWS.

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1910 Dr. Alex. Graham Bell, 1331 Connecticut Ave., N. W. Washington, D. C.
1907 Dr. Guis Grandenigo, Via Guglielmo Sanfelice, 33 Naples, Italy
1900 Sir James Dundas-Grant, 148 Harley St., W. I. London, Eng.
1903 Dr. Edward Law, 8 Wimpole St., Cavendish Square, London, Eng.
1919 Dr. Fernand Lemaitre.....6 Sq. Moncy, Paris, France
1909 Dr. James Kerr Love, 650 Shields Doad, Pollokshields, Glasgow, Scotland
1896 Dr. H. Luc.....54 Rue de Varenne, Paris, France
1896 Dr. John MacIntyre.....179 Bath St., Glasgow, Scotland
1898 Dr. E. J. Moure, 25 Cours du Jardin Public, Bordeaux France
1919 Dr. Frank Rose.....68 Wimpole St., London, W., Eng.
1898 Dr. E. Schmieglow, 18 Norregade, Copenhagen, Denmark
1898 Dr. J. Sendziak.....10 Crywariska St., Warsaw, Poland
1919 Sir St. Clair Thomson, 64 Wimpole St., London, W., Eng.
1902 Dr. Logan Turner....27 Walker St., Edinburgh, Scotland

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1920	Wells P. Eagleton, M. D.	Joseph B. Greene, M. D.	Samuel Iglauer, M. D.	H. Bert Ellis, M. D.
1921	Harmon Smith, M. D.	Joseph B. Greene, M. D.	Joseph C. Beck, M. D.	Hill Hastings, M. D.
1922	J. F. Fairbairn, M. D.	T. C. Worthington, M. D.	Greenfield Sluder, M. D.	Grant Selfridge, M. D.
1923	F. Nolton Bigelow, M. D.	W. G. Harrison, M. D.	Gordon B. New, M. D.	Isaac H. Jones, M. D

Mid-Western Section.

1918	Frank R. Spencer, M. D.	1921	William V. Mullin, M. D.
1919	Frank L. Dennis, M. D.	1922	J. M. Foster, M. D.
1920	Claude E. Cooper, M. D.	1923	Robert Levy, M. D.

SECRETARY AND TREASURER.

1896 to 1900
Robert Cunningham Myles, M. D.
1901 to 1906
Wendell C. Phillips, M. D.
1907 to 1916
Thomas J. Harris, M. D.
1917 to date
William H. Haskin, M. D.

1901 to 1907
Ewing W. Day, M. D.

1908 Chevalier Jackson, M. D.

1909 to date
Ewing W. Day, M. D.

COUNCIL.

1896—T. P. Berens, M. D.
J. H. Billings, M. D.
D. L. Hubbard, M. D.
H. H. Curtis, M. D.
W. C. Phillips, M. D.
R. C. Myles, M. D.
1897—G. H. Makuen, M. D.
T. P. Berens, M. D.
W. C. Phillips, M. D.
H. H. Curtis, M. D.
E. W. Day, M. D.
D. L. Hubbard, M. D.
1898—J. E. Nichols, M. D.
H. H. Curtis, M. D.
L. A. Coffin, M. D.
W. C. Phillips, M. D.
E. W. Day, M. D.
C. W. Richardson, M. D.
S. E. Solly, M. D.
1899—W. H. Daly, M. D.
W. C. Phillips, M. D.
T. H. Halstead, M. D.
J. O. Roe, M. D.
H. W. Loeb, M. D.
T. C. Christy, M. D.
F. L. Jack, M. D.
D. L. Hubbard, M. D.
1900—S. E. Solly, M. D.
W. H. Daley, M. D.
A. W. Calhoun, M. D.
J. B. Clemens, M. D.
J. F. McKernon, M. D.
W. C. Phillips, M. D.
J. E. Sheppard, M. D.
J. Price Brown, M. D.
Max Thorner, M. D.
1901—D. B. Kyle, M. D.
S. MacCuen Smith, M. D.
S. E. Solly, M. D.
J. Price Brown, M. D.
W. H. Daley, M. D.
J. B. Clemens, M. D.
J. F. McKernon, M. D.
C. W. Richardson, M. D.
F. C. Cobb, M. D.
1902—D. B. Kyle, M. D.
S. E. Solly, M. D.
J. F. McKernon, M. D.
S. MacCuen Smith, M. D.
F. C. Cobb, M. D.
N. H. Pierce, M. D.
T. P. Berens, M. D.
J. A. White, M. D.
1903—C. W. Richardson, M. D.
R. C. Myles, M. D.
D. B. Kyle, M. D.
J. F. McKernon, M. D.
S. MacCuen Smith, M. D.
F. C. Cobb, M. D.
N. H. Pierce, M. D.
T. P. Berens, M. D.
C. R. Holmes, M. D.
1904—J. A. Stucky, M. D.
C. W. Richardson, M. D.
R. C. Myles, M. D.
A. G. Root, M. D.
T. P. Berens, M. D.
C. R. Holmes, M. D.
E. B. Dench, M. D.
F. L. Jack, M. D.
J. E. Schadle, M. D.
F. R. Packard, M. D.
1905—N. H. Pierce, M. D.
J. A. Stucky, M. D.
C. W. Richardson, M. D.
L. A. Coffin, M. D.

T. H. Halsted, M. D
C. R. Holmes, M. D.
E B. Dench, M. D.
F. L. Jack, M. D.
J. E. Schadle, M. D.
F. R. Packard, M. D.

1906—F. C. Cobb, M. D.
N. H. Pierce, M. D.
J. A. Stucky, M. D.
E. B. Dench, M. D.
F. L. Jack, M. D
F. R. Packard, M. D.
L. A. Coffin, M. D.
T. H. Halsted, M. D.
J. F. McKernon, M. D.
H. W. Loeb, M. D.

1907—J. E. Logan, M. D.
F. C. Cobb, M. D.
N. H. Pierce, M. D.
L. A. Coffin, M. D.
T. H. Halsted, M. D.
J F. McKernon, M. D.
H. W. Loeb, M. D.
C. W. Richardson, M. D.
D. B. Kyle, M. D.

1908—W. C. Phillips, M. D.
J. E. Logan, M. D.
F. C. Cobb, M. D.
J. F. McKernon, M. D.
H. W. Loeb, M. D.
C. W. Richardson, M. D.
D. B. Kyle, M. D.
C. R. Holmes, M. D.
J. M. McCaw, M. D.

1909—J. E. Logan, M. D.
D. B. Kyle, M. D.
C. W. Richardson, M. D.
W. C. Phillips, M. D
J. M. McCaw, M. D.
W. R. Lincoln, M. D.
G. L. Richards, M. D.
J. F. Barnhill, M. D.
G. H. Makuen, M. D.

1910—C. R. Holmes, M. D.
G. L. Richards, M. D.
J. F. Barnhill, M. D
G. H. Makuen, M. D.
N. H. Pierce, M. D.
W. C. Phillips, M. D.
F. R. Packard, M. D.
A. B. Duel, M. D.
Robert Levy, M. D.

1911—J. F. McKernon, M. D.
J. A. White, M. D.
N. L. Wilson, M. D.
C. R. Holmes, M. D.
A. B. Duel, M. D.
F. R. Packard, M. D.
Robert Levy, M. D.
G. L. Richards, M. D.
N. H. Pierce, M. D.

1912—C. R. Holmes, M. D.
F. R. Packard, M. D.
N. H. Pierce, M. D.
J. F. McKernon, M. D.
J. A. White, M. D.
N. L. Wilson, M. D.
C. Jackson, M. D.
M. A. Goldstein, M. D.
A. B. Duel, M. D.

1913—James F. McKernon, M. D.
Joseph A. White, M. D.
Norton L. Wilson, M. D.
Chevalier Jackson, M. D.
Max A. Goldstein, M. D.
Arthur B. Duel, M. D.
Harris P. Mosher, M. D.
E. Fletcher Ingals, M. D.
G. Hudson-Makuen, M. D.

1914—Chevalier Jackson, M. D.
Max A. Goldstein, M. D.
Arthur B. Duel, M. D.
Harris P. Mosher, M. D.
E. Fletcher Ingals, M. D.
G. Hudson-Makuen, M. D.
H. Holbrook Curtis, M. D.
John E. Sheppard, M. D.
Robert Levy, M. D.

1915—Joseph A. White, M. D.
Harris P. Mosher, M. D.
E. Fletcher Ingals, M. D.
G. Hudson-Makuen, M. D.
H. Holbrook Curtis, M. D.
John E. Sheppard, M. D.
Robert Milligan, M. D.
John M. Ingersoll, M. D.
B. R. Shurley, M. D.

1916—G. Hudson-Makuen, M. D.
H. Holbrook Curtis, M. D.
John E. Sheppard, M. D.

Robert Milligan, M. D.
John M. Ingersoll, M. D.
B. R. Shurley, M. D.
E. B. Dench, M. D.
D. J. Gibb Wishart, M. D.
Robert Levy, M. D.

1917—Robert Milligan, M. D.
John M. Ingersoll, M. D.
E. B. Dench, M. D.
D. J. Gibb Wishart, M. D.
B. R. Shurley, M. D.
Robert Levy, M. D.
S. MacCuen Smith, M. D.
Thomas H. Halsted, M. D.
Walter A. Wells, M. D.

1918—D. J. Gibb Wishart, M. D.
B. R. Shurley, M. D.
Robert Levy, M. D.
S. MacCuen Smith, M. D.
Thomas H. Halsted, M. D.
Walter A. Wells, M. D.
T. J. Harris, M. D.
Francis P. Emerson, M. D.
L. W. Dean, M. D.

1919—S. MacCuen Smith, M. D.
Thomas H. Halsted, M. D.
Walter A. Wells, M. D.
Thomas J. Harris, M. D.
Francis P. Emerson, M. D.
L. W. Dean, M. D.
Frank R. Spencer, M. D.
Joseph C. Beck, M. D.
Wendell C. Phillips, M. D.

1920—Class A
Thomas J. Harris, M. D.
Lee Wallace Dean, M. D.
Francis P. Emerson, M. D.
Class B
George L. Richards, M. D.
Joseph C. Beck, M. D.
Frank R. Spencer, M. D.
Wendell C. Phillips, M. D.
Class C
Herbert S. Birkett, M. D.
LaFayette Page, M. D.
Robert C. Lynch, M. D.

1921—Class A
George L. Richards, M. D.
George E. Shambaugh, M. D.
Frank R. Spencer, M. D.
Wendell C. Phillips, M. D.
Class B
Herbert S. Birkett, M. D.
LaFayette Page, M. D.
Robert C. Lynch, M. D.
Class C
Harris P. Mosher, M. D.
Joseph F. McCaw, M. D.
Dunbar Roy, M. D.

1922—Class A
Herbert S. Birkett, M. D.
LaFayette Page, M. D.
Robert C. Lynch, M. D.
Class B
Harris P. Mosher, M. D.
Joseph F. McCaw, M. D.
Dunbar Roy, M. D.
Class C
Lee Wallace Dean, M. D.
James F. McKernon, M. D.
Norval H. Pierce, M. D.

1923—Class A
Harris P. Mosher, M. D.
J. T. McCaw, M. D.
J. W. Jervey, M. D.
Class B
Lee Wallace Dean, M. D.
J. F. McKernon, M. D.
N. H. Pierce, M. D.
Class C
T. A. Coffin, M. D.
B. R. Shurley, M. D.
R. H. Skillern, M. D.

DECEASED WHILE MEMBERS OF THE SOCIETY.

Resident Members.

Noyes, J. L., M. D.....1896
O'Dwyer, Joseph, M. D.....1897
Hengst, D. A., M. D.....1898
Nichols, J. E. H., M. D.....1898
Hopkins, Woolsey, M. D.....1899
Rankin, D., M. D.....1900
Wenner, R. J., M. D.....1900
Chapman, S. Hartwell, M. D.....1903

Friedenberg, Edward, M. D.....1903
Kibbe, A. B., M. D.....1903
Hoople, H. Nelson, M. D.....1905
Brandegee, Wm. P., M. D.....1906
Solly, S. E., M. D.....1906
Harland, W. G. B., M. D.....1907
Burnett, Peter V., M. D.....1908
Roosa, D. B., St. John, M. D.....1908
Schadle, J. E., M. D.....1908
Pette, C. H., M. D.....
Sprague, F. B., M. D.....1909
Mullins, John B., M. D.....1909
McGahan, C. F., M. D.....1910
Anderson, Willis S., M. D.....1910
Calhoun, A. W., M. D.....1910
Gradle, Henry, M. D.....1911
Koyle, F. H., M. D.....1911
Dudley, Wm. F., M. D.....1912
Kimball, Irving E., M. D.....1913
Gildea, Patrick F., M. D.....1913
Adams, J. L., M. D.....1914
Gibb, Jos. S., M. D.....1914
Gruening, Emil, M. D.....1914
Sheppard, John E.....1916
Roe, John O.....1916
Ballenger, Wm. L.....1916
Dye, Hobart S.....1917
Kyle, D. Braden.....1917
Makuen, G. Hudson.....1917
Solenberger, Amos R.....1917
Daley, Walter S.....1918
Ingals, E. Fletcher.....1918
Yates, D. G.....1918
Chappell, Walter F.....1919
Ray, James Morrison.....1919
Bird, N. S.....1919
Blake, Clarence J.....1919
Holmes, E. M.....1919
Holmes, Christian R.....1920
Horn, Henry W.....1920
Lutz, Stephen Henry.....1920
Kenefick, Joseph Aloysius.....1920
Friedberg, S. A.....1920
Curtis, H. Holbrook.....1920
Cort, Geo. F.....1921
Linhart, C. P.....1921
Kyle, J. J.....1921
Kuyk, D. A.....1922
Lynah, H. L.....1922

HONORARY MEMBERS WHO HAVE DIED.

Whistler, W. McNeil.....1899
Gougenheim, Achille.....1901
Gruber, Joseph.....1901
Brown, Lenox.....1902
Smyly, Sir Philip.....1904
Joal, Joseph.....
Woakes, Edward.....
Semon, Sir Felix.....1921

EMERITUS FELLOWS WHO HAVE DIED.

Ellis, H. Bert.....1927

RF
1
A53
1922

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